

**PAGE NOT  
AVAILABLE**













REVIEW  
OF  
NEUROLOGY AND PSYCHIATRY

EDITOR

ALEXANDER BRUCE  
M.D., F.R.C.P.E., F.R.S.E.

ASSISTANT EDITORS

EDWIN BRAMWELL  
M.B., F.R.C.P.E., F.R.S.E., M.R.C.P.LOND.

CHAS. MACFIE CAMPBELL  
M.B., CH.B.

VOLUME IV.



EDINBURGH  
OTTO SCHULZE & COMPANY  
20 SOUTH FREDERICK STREET

1906

RC321

R4

v. 4

12/1/11

EDINBURGH

*Printed by*

TURNBULL & SPEARS

12/1/11

# Contents

## ORIGINAL ARTICLES.

	PAGE
THE INFLUENCE OF FACIAL HEMIATROPHY ON THE FACIAL AND OTHER NERVES. By Sir William Gowers, M.D., F.R.S. . . . .	1
A SECOND CASE OF PARTIAL DOUBLING OF THE SPINAL CORD. By Alexander Bruce, M.D., F.R.C.P.E.; Stuart McDonald, M.B., F.R.C.P.E.; and J. H. Harvey Pirie, B.Sc., M.B., Ch.B. . . . .	6
PARAMYOCLONUS EPILEPTICUS. By Ernest S. Reynolds, M.D. (Lond.), F.R.C.P. . . . .	19
LESIONS OF THE SPINAL CORD, THE RESULT OF ABSORPTION FROM LOCALISED SEPTIC FOCI, WITH A PRELIMINARY NOTE ON AN EXPERIMENTAL RESEARCH. By David Orr, M.D., and R. G. Rows, M.D. . . . .	25
THE PATHOLOGY OF GENERAL PARALYSIS OF THE INSANE. By W. Ford Robertson, M.D. . . . .	73, 169, 258
A STUDY OF SOME CASES OF DELIRIUM PRODUCED BY DRUGS. By August Hoch, M.D. . . . .	83
THE HISTOLOGICAL APPEARANCES OF THE CORD AND MEDULLA IN A CASE OF ACUTE ASCENDING PARALYSIS. By Charles Workman, M.D., and Walter K. Hunter, M.D., D.Sc. . . . .	106
TRYPANOSOMIASIS OR SLEEPING SICKNESS. By D. G. Marshall, Major, I.M.S. (Retd.) . . . . .	112
A CASE OF CHRONIC PROGRESSIVE DOUBLE HEMIPLEGIA. By E. Farquhar Buzzard, M.D., M.R.C.P., and Stanley Barnes, M.D., M.R.C.P. . . . .	182
A CASE OF MUSCULAR DYSTROPHY AFFECTING HANDS AND FEET: DEPRESSION AFTER EXHAUSTION, WITH RECOVERY. By C. Macfie Campbell, M.B., Ch.B. . . . .	192
ON TETANOID CHOREA AND ITS ASSOCIATION WITH CIRRHOSIS OF THE Liver. By Sir W. R. Gowers, M.D., F.R.S. . . . .	249
A CASE OF CYSTICERCUS CELLULOSE OCCURRING IN THE INSANE. By H. Egerton Brown, M.D. . . . .	272
LESIONS OF THE LEFT FIRST TEMPORAL CONVOLUTION IN RELATION TO SENSORY APHASIA. By William G. Spiller, M.D. . . . .	329



	PAGE
TWO CASES OF EMBRYOMA IN THE FRONTAL LOBE OF THE BRAIN. By R. G. Rows, M.D. . . . .	338
THE RECOGNITION OF SEGMENTAL LEVELS IN THE CERVICAL AND LUMBAR ENLARGEMENTS OF THE SPINAL CORD FROM THE APPEARANCE OF THE TRANSVERSE SECTION. By Edwin Bram- well, M.B., F.R.C.P.E., M.R.C.P. Lond. . . . .	344
THE PATHOLOGY OF A CASE OF MYELITIS ACUTISSIMA HÆMORR- HAGICA DISSEMINATA. By W. B. Warrington, M.D., F.R.C.P., and John Owen, M.B. . . . .	401
A CASE OF HÆMORRHAGE INTO THE BRAIN AND SPINAL CORD FROM OBLITERATIVE ARTERIAL DISEASE. By W. B. Warrington, M.D., F.R.C.P., and John Owen, M.B. . . . .	407
THE PARS INTERMEDIA OR NERVUS INTERMEDIUS OF WRISBERG, AND THE BULBO-PONTINE GUSTATORY NUCLEUS IN MAN. By Dr J. Nageotte . . . . .	473
THE DESCENDING DEGENERATIONS OF THE POSTERIOR COLUMNS IN (1) TRANSVERSE MYELITIS, AND (2) AFTER COMPRESSION OF THE DORSAL POSTERIOR ROOTS BY TUMOURS. By David Orr, M.D. . . . .	488
THE PATHOLOGY OF GENERAL PARALYSIS. By Dr Hans Evensen	537, 616
ON THE FREQUENCY WITH WHICH CERTAIN SIGNS AND SYMPTOMS OCCUR IN CASES OF DISSEMINATED SCLEROSIS BEFORE THE DEVELOPMENT OF SO-CALLED CARDINAL SIGNS. By Ashley W. Mackintosh, M.A., M.D. . . . .	601
PRECOCIOUS PARALYSIS OF THE PALATE IN DIPHTHERIA. By J. D. Rolleston, M.A., M.D. (Oxon.) . . . . .	608
TYPES OF THE DEVOLUTIONAL PSYCHOSES. By Clarence Farrar, M.D.	665
A CASE OF PARTIAL DOUBLING OF THE SPINAL CORD. By Purves Stewart, M.A., M.D. Edin., F.R.C.P.; and Julius Bernstein, M.B. Lond., M.R.C.S. . . . .	729
A NOTE UPON TWO IMPORTANT POINTS IN THE LOCALISATION OF TUMOURS OF THE FRONTAL REGION OF THE BRAIN. By T. Grainger Stewart, M.B. Ed., M.R.C.P. Lond. . . . .	809
NOTE ON A CASE OF JUVENILE GENERAL PARALYSIS; ABSENCE OF STIGMATA OF CONGENITAL SYPHILIS AND OF A FAMILY HISTORY INDICATIVE OF THAT DISEASE; VERY PRONOUNCED CEREBRO-SPINAL LYMPHOCYTOSIS. By Edwin Bramwell, M.B., F.R.C.P.E., F.R.S.E., M.R.C.P. Lond. . . . .	813

# **Review**

of

# **Neurology and Psychiatry**

---

## **Original Articles**

### **THE INFLUENCE OF FACIAL HEMIATROPHY ON THE FACIAL AND OTHER NERVES**

By Sir W. R. GOWERS, M.D., F.R.S.

IN facial hemiatrophy the temporal bone may share the diminution in size of the bones of the face, and this is shown by the smaller mastoid process, and also by the smaller external auditory meatus. The latter fact is of significance, because we cannot doubt that other bony canals also become narrowed, and among them the Fallopian canal through which the facial nerve passes. We can therefore understand that this nerve may suffer in facial hemiatrophy, as in the three cases here described. Contraction of the canal may act directly or indirectly. Manifestly a much slighter degree of inflammatory swelling of the sheath of the nerve would impair its function than would be necessary in a canal of normal size. The diminution in size may also alter the blood-supply in the sheath and thus dispose to vascular disturbance. It may conceivably also cause some direct mechanical pressure or irritation.

Facial hemiatrophy is so rare, that it is impossible that its connection with the affection of the facial nerve in the following cases can be an accidental one.

**CASE I.** The first case is that of a young man, 25 years of age, in whom left-sided facial paralysis came on six years before I  
R. OF N. & P. VOL. IV. NO. 1.—A

saw him. It was thought to be due to exposure to cold, but no clear history of the onset could be obtained. There was considerable paralysis of all parts of the face, and he still could not quite close the eyelids, but late contraction had restored the symmetry of the face when at rest. There were slight flickering contractions in the orbicularis palpebrarum. The electrical excitability of the nerve and muscle had become that which is common in old cases; in both it was much reduced to each current, but nowhere was it extinct.

There was distinct atrophy of the bones of the left side of the face, the frontal, superior maxillary, and malar bones. The mastoid process was also markedly smaller than the right, and the auditory meatus was so much less that it would hardly admit the smallest speculum, while into the right a full-sized one could be readily introduced.

The facial hemiatrophy had not been recognised before. The condition of the facial muscles was that which would be found from a severe neuritis after the time which had elapsed. But if so, the condition of the canal must be regarded as having an influence, especially in view of the remarkable features of other cases.

CASE II. A single woman, aged 37, suffered from spasms in the right side of the face, which had commenced three years before. At first it was slight and seldom, but had gradually become greater and practically constant. There was distinctly less movement on the right side. The naso-labial furrow was much less on the right side, and there was less power in the zygomatic muscles. Between the movements in the upper part of the face on the two sides no difference could be seen. There were frequent short attacks of clonic spasm in the right orbicularis, occasionally spreading to the cheek. Movement did not excite them, except contraction of the frontalis, which always brought on the spasm in the orbicularis. Sensation in the face, and power in the masseter, were normal. The bones of the right face were distinctly smaller than in the left. The difference was least in the lower jaw, but even here was distinct. It was marked in the superior maxillary and malar bones, and also in the mastoid process. Moreover, the external auditory meatus was distinctly smaller than that on the left side.

Hearing was quite good, but slight tinnitus was present. Nine months later her condition was almost the same. There was the same frequent spasm in the orbicularis, but that in the cheek seemed to be chiefly in the elevator of the upper lip, occasionally spreading to the zygomatic muscles. The tinnitus persisted, and was described as a humming, which seemed to be felt as well as heard.

In this patient, also, the symptoms of slight interference with the facial nerve were associated with the diminution in size of the bones. In each case, advice was sought for the symptoms in the face; in neither had the alteration in the bones been previously observed. Whatever may be the relation between the two, the cases show the great importance of a careful observation of the bones in all cases of facial paralysis and spasm.

CASE III. The last case is still more remarkable, because other nerves suffered as well as the facial.

A solicitor, aged 42, came to me in 1903 on account of left-sided facial paralysis which had come on two years before. The precise onset could not be ascertained, but the loss of power was probably not complete and not sudden. It was said to be accompanied by some chronic twitching of the eyelids. When seen, there was moderate loss of power in all parts of the face, but nowhere complete paralysis. There was distinct late overaction, causing slight contraction of the zygomatic muscles, and undue approximation of the eyelids in a smile. Indeed there was a slight narrowing of the palpebral fissure at rest. He was sent to me because he was thought to have ptosis as well as facial paralysis, but the supposed ptosis was really due to the slight contraction of the orbicularis, and also to the fact that the left eye-ball and orbit were a little lower in position than the right. This was due to distinct left hemiatrophy. All bones of the face were definitely smaller on the left side than on the right, including the lower jaw and the frontal bone. The condition had not been recognised, and it was impossible to ascertain when it had come on. But I had seen him on two occasions before; first fifteen years, and the second time six years before, each time on account of troublesome general headache, to which he had long been liable. On each occasion I examined him carefully, lest

there should be any organic disease, and I cannot think I should have passed unnoticed such a condition. Moreover, on the last occasion (1897) he complained that he could not hear so well with the left ear, but no loss of hearing was present, even to high notes. In 1903, however, he had lost all hearing on the left side, evidence of a progressive process. Sensation on the face was normal; so were the masseter and the ocular movements; the left side of the palate was weak, and the vocal cord was found by Sir Felix Semon to be moved much less than the other. In addition to the atrophy of the bones of the face, the left mastoid process was smaller than the right and the external auditory meatus considerably reduced in size.

I saw him again a year later (1904). In spite of treatment the same condition continued. The face was not worse, the deafness remained complete, sensation in the face was not impaired, but the masseter seemed a little weaker than the right. The left side of the palate had become completely paralysed, and so had the left vocal cord. At times he had some difficulty of swallowing, lasting an hour or two. His pulse was persistently 116-120. He frequently woke up at night with a sense of great dyspnoea. Moreover, there was some wasting of the sterno-mastoid and trapezius on the left side.

Once more I saw him, a few months later, but he was then dying of pneumonia, which had come on most acutely, from slight exposure during a trifling attack of influenza. The pneumonia was in the left lung, but was beginning to affect the other, and the heart was rapidly failing. He died a few hours after the signs of pneumonia were distinct. There had been no marked change in his symptoms.

The nerve palsies in this case clearly point to narrowing, not only of the canal for the facial nerve, but also of the foramen lacerum. Through this pass the spinal accessory nerve (which is known to supply the palate as well as the vocal cord) and also the pneumo-gastric and glosso-pharyngeal. Of these, the first was clearly paralysed and the others were apparently impaired, and may have promoted his death. I have seen similar symptoms (including the paroxysmal dyspnoea and frequency of the heart's action) from a growth in the temporal bone. In this patient a growth can certainly be excluded.

It may be thought that a shrinkage of the temporal bone

would enlarge the foramen lacerum, and not narrow it, since it is situated between the temporal and the occipital bones. But in the disease we call "facial hemiatrophy" the reduction in size is not of the bones; it is of the bone. It ignores the sutures, and never opens them. If the shrinkage of the temporal bone, conspicuous in the smaller size of the mastoid process, reached the junction with the occipital bone, we should expect also the adjacent part of this to have suffered, and thus the foramen lacerum may have been lessened in size. Unfortunately I omitted to observe the condition of the bone behind the mastoid process. The foramen is somewhat crossed by bone, separating the part occupied by the nerves from the rest. In this way their compression may possibly have been facilitated.<sup>1</sup>

It is probable that more than one morbid state is included under the term "facial hemiatrophy." It is a misnomer applied to such cases as these, especially to the last, in which the bones seemed to have suffered through a large extent of one half of the cranium. The condition was "facial" only because the general asymmetry was most readily perceived in the face.

A remarkable fact is the probability that the state was a real atrophy, and not an arrested development. Not only was the condition unnoticed six years before, but during this time slight deafness became complete, and the first symptom of impairment of other nerves was noticed less than four years before death.

It is to be regretted that the pathological condition could not be ascertained, and that the symptoms could not be more minutely observed and closely watched. But it is well known how difficult it is to secure scientific facts from cases only seen in private.

<sup>1</sup> Prof. Thane has given me particulars of one instance in which a broad piece of bone, from the occipital, passed between the jugular part of the foramen and that for the nerves.

## **A SECOND CASE OF PARTIAL DOUBLING OF THE SPINAL CORD.**

By ALEXANDER BRUCE, M.D., F.R.C.P.E., Physician to the  
Royal Infirmary, Edinburgh ;

STUART M'DONALD, M.B., F.R.C.P.E., Pathologist to the Royal Infirmary ;  
and

J. H. HARVEY PIRIE, B.Sc., M.B., Ch.B., Clinical Tutor in the  
Royal Infirmary.

THE cord was obtained from a patient, M. A., who died in June 1904, at the age of fifty years, from exhaustion and asphyxia caused by a malignant growth arising from a bronchus in the right lung, and involving the superior mediastinum as well as the lung itself. From October 1902 she had suffered from pain and swelling in the right leg below the knee, and in September 1903 the leg was amputated just above the knee, as the swelling was found to be malignant. At the post-mortem examination it was found that there were also some areas of softening in the cerebrum, but nothing abnormal was noted about the spinal cord, spinal membranes, or vertebral column.

The woman had been a dressmaker by occupation, and there was nothing in her previous history or in her family history to indicate any abnormality of the spinal cord. The cord was being examined with a view to the localisation of the motor nuclei of the leg muscles when it was discovered that there was a partial double formation extending over the upper four sacral segments.

The cord had been fixed in formalin, divided into segments, embedded in celloidin, and cut into serial sections. For the purposes of this examination every tenth section was further hardened in Müller's fluid and then stained by the Weigert-Pal method.

The first change is seen in the lower half of L 5, where the central canal becomes elongated in an antero-posterior direction,

extending backwards almost to the periphery in the line of the posterior median septum. The posterior columns are also very wide. In the upper third of S 1 this posterior elongation persists, but it now lies in a narrow wedge of grey matter. This wedge gradually increases in width; the central canal divides into two, one division being in the normal situation and somewhat dilated, the other lying in the middle line near the posterior periphery. This latter soon ends blindly; the former then becomes elongated transversely, and a little below the middle of the segment divides into two canals lying in the same frontal plane. The anterior median fissure becomes  $\Lambda$ -shaped and persists so till near the lower end of S 4, one central canal lying opposite the extremity of each limb of the  $\Lambda$ .

At the level of the lower division of the central canal the median posterior wedge of grey matter is distinctly recognisable as posterior cornu through the presence of gelatinous substance. Before the lower end of the segment is reached it is being split into two cornua by the intervention of a median band of white fibres.

In S 2, although in outward shape still apparently a single cord, its double nature is more evident. The two outer anterior and posterior cornua, representing the continuations downwards of the normal grey matter at higher levels, are (except for the group of cells on the right side in degenerative reaction) normal.

The two inner posterior cornua are perfectly formed, although nerve roots cannot with certainty be traced into them. On their outer side there are indications of posterior columns separated by posterior "median" septa from those of the corresponding outer halves. Between them is white matter (lateral columns?) somewhat split up by fissures—attempts at division of the double cord into separate entities. There are two rudimentary inner anterior cornua in contact in the upper part of the segment, but almost separated below by white matter which runs uninterruptedly from posterior periphery to anterior median fissure.

In S 3 the fissures (containing pia mater) in the white matter between the two inner posterior cornua reach as far forwards as the inner anterior cornua, but never succeed in entirely dividing the cord into two parts. The inner anterior



cornua are still very rudimentary, but, especially on the left side, there are a few large, well-formed motor cells, with nerve roots arising from them, and reaching the surface through the anterior median fissure. No posterior nerve roots are seen entering the inner posterior cornua.

In the upper half of S 4 the inner anterior cornua gradually diminish in size and finally disappear. The white matter between the inner posterior cornua shrinks till they come into contact. The anterior median fissure is still  $\Lambda$ -shaped; there are two central canals and two posterior "median" septa, one on each outer aspect of the inner posterior cornua.

In the lower half of the segment the central canals gradually approach and ultimately join into a single, truly central one; the median wedge of grey matter (fused inner posterior cornua) becomes cut off in front from the grey commissure by white matter (normal posterior columns), and gradually diminishes in width until it becomes a mere strand in the line of the single posterior median septum. This finally disappears, and at the extreme lower end of the segment we have an absolutely normal cord.

At first this duplication resembled very closely that of Theodor's case in the appearance and gradual growth of the posterior median wedge of grey matter; as also in the presence of two central canals and four anterior and four posterior horns, while the two cords were still connected. In Theodor's case, however, the two cords became separated, while in ours they always remained united, and before the lower end of the cord was reached the conditions were again normal, the return to the single condition being effected in a manner closely resembling that in which the transformation to a double condition was carried out.

Steiner has collected thirty-five cases and reported an additional one of his own. As these have been published only in the form of a thesis and are not, therefore, generally accessible, we place here a short synopsis of them.

## SYNOPSIS OF STEINER'S CASES.

NAME	AGE	PART OF CORD	CONDITION OF VERTEBRÆ AND MEMBRANES	CONDITION OF CORD
(1) Zacchias	(Anencephalus fœtus)	—	"Caput a postica parte, carne seu pelle erat detectum"	"Qua etiam medulla spinalis erat denuta, quæ duplex conspiciebatur."
(2) Manget (1695)	(Anencephalus 9 months' fœtus)	—	—	"Amplior multo quam in aliis fœtibus et bifida ad oesim usque sacri limina conspiciebatur."
(3) Hull	—	—	—	Cord appeared in form of two slender cords, each giving rise to nerves.
(4) Ollivier	(Anencephalus)	—	—	Cord consisted of two small white threads, slightly rounded posteriorly, flattened anteriorly, close to each other, very straight, about the size of a crow's quill.
(5) Sandifort	—	Lumbar region	Meningocele	Cord split only by a "sulcus non admodum profundus."
(6) Grashuy (cited by Recklinghausen)	—	1st sacral	Sacral hydromeningocele. Splitting of vertebral arch of the os sacrum	Cord split in 1st sacral region, each half being displaced laterally.
(7) Natorp	—	7th cervical to 5th dorsal	A splitting in the spinal processes from the 7th cervical and 1st dorsal as far as the 5th dorsal	Cord split ( <i>but not doubled</i> ).
(8) Cruveilhier	—	12th dorsal, 1st and 2nd lumbar	Splitting through vertebral processes from D 12 to L 5, and bodies from D 12 to L 2	Cord divided into two columns by a bony process from the 12th dorsal vertebra; each half has a wide central canal. Duplication extends as high as 4th ventricle.
(9) Ammon	Fœtus	Cervical and upper dorsal	"Fissura spinalis totalis"	Cord broader than normal, and divided into two parts, each containing a fine canal which can be followed into the 4th ventricle.
(10) Tarum (cited by Recklinghausen)	—	—	Spina bifida	Division of cord, caused by the right arch of the lumbar vertebra, which, instead of joining with its fellow, reaches into the middle of the spinal canal. ( <i>Merely a splitting of cord.</i> )

SYNOPSIS OF STEINER'S CASES—*continued.*

NAME	AGE	PART OF CORD	CONDITION OF VERTEBRÆ AND MEMBRANES	CONDITION OF CORD
(11) Lenhossék	6 months' fetus	Lumbar enlargement	No doubling of vertebræ	Two lumbar enlargements, with two cords; left fully developed, right representing only right half of a cord. No doubling above or below. Three anterior and three posterior roots.
(12) Öllacher	Chick embryo of 4 days	Dorsal region	—	Medullary tube split into several parts, normal above and below.
(13) Foà	Man, 76	Lumbar region	Vertebral canal intact	Cord split for 2 cm. into a right slender and a left broad half. Central canal broadened above splitting. Doubled in region of split. In both halves H-formed grey matter.
(14) Fürstner and Zacher	50	As far down as lowest dorsal	Vertebræ and membranes normal	Inside same pial sac was a second fully developed cord, which gradually compressed the first, and finally took its place. In lowest lumbar, cord again single.
(15) Recklinghausen	Woman, 31	Lumbar region	No changes in vertebræ	Cord for 9 cm. divided into two cylindrical columns, surrounded by pia, having two anterior and two posterior horns each. Inner halves less perfect than outer. (Figures.)
(16) Recklinghausen	(Anencephalus) fetus	Lumbar region	Bony process from 1st lumbar vertebra	Bony process from 1st lumbar penetrating into cord, and splitting it for 15 cm. (Cord split, not doubled.)
(17) Recklinghausen	—	"Area medullo-vascular"	(Spina bifida) bony process from lower layer of skin, through dura into cord. (Not connected with vertebra)	Cord for 2.5 cm. split by bony process into a right and a left cylindrical column, 3 mm. thick; these join again into a small conus. (Cord split, not doubled.)
(18-21) Recklinghausen	Fœtuses	—	4 cases of spina bifida	Signs of lateral division of cord rudiments. (Cord split, not doubled.)
(22) Beneke	—	—	—	Cord split into two asymmetrical halves, by intrusion of a fold of membrane. (Probably a mere splitting of cord.)

SYNOPSIS OF STEINER'S CASES—*continued*.

NAME	AGE	PART OF CORD	CONDITION OF VERTEBRÆ AND MEMBRANES	CONDITION OF CORD
(23) Bonome	2 years	Lower lumbar	Split through dura. (No spina bifida)	Cord split by layer of connective tissue. Small piece of cartilage in septum of connective tissue. Splitting commences at periphery of Goll's column, where 2nd pair of posterior horns appears. Gradually two cords form, their anterior horns medially, their posterior horns laterally.
(24) Miura	53 (trauma)	Dorsal region. Doubling from 2nd lumbar	Cystoid cavity communicating with central canal.	Cord doubled from 2nd lumbar; central canal divided; 2nd inner pairs of well-formed posterior horns; lower down rudimentary pair of anterior horns. Gradually two central canals join, and inner pair of posterior horns disappear (Figures).
(25, 26) Fischer	(2 cases)	Lower cervical and lumbar regions	Displacement of vertebræ; (in one case penetration into central canal)	Splitting of cord into two halves, which spread out membrane-like in region of the spina bifida.
(27) Kronthal	In an ox	—	Cord and membranes had been cut by a butcher's axe	Partial doubling of cord. Along with a transverse section almost normal, there was a section of another half cord.
(28) Rosenberg	3 years	Middle dorsal	Spina bifida.	Splitting, with double central canal. Lower down cord diminishes into a membrane-like mass, and still lower again increases in circumference.
(29) Steffen	6 months	5th lumbar	Fissure in 5th lumbar. Injury to bone, divided vertebral canal	Cord divided by injury.
(30) Jakobsch	65	Lumbar enlargement	—	In lumbar enlargement, at left side, a formation like a sand-glass, connected with medullary substance.
(31) Feist	—	Dorsal and lumbar regions	—	A secondary cord at ventral surface, between dorsal and lumbar regions. Had all the form of a normal cord. Further up the two pairs of anterior horns again unite.
(32) Brasch	49	Upper dorsal	Vertebræ normal	In grey matter a peculiar bundle of white substance, and an extrapial formation of grey substance, which latter is described as a rudimentary partial doubling of the cord.

SYNOPSIS OF STEINER'S CASES—*continued.*

NAME	AGE	PART OF CORD	CONDITION OF VERTEBRÆ AND MEMBRANES	CONDITION OF CORD
(33) Turner	Rabbit	—	—	Cord with two canals, an accessory anterior horn, and three anterior nerve roots.
(34) Chiari	17	Lumbar	Myelo-meningocele.	Total splitting of cord. Nervous elements well preserved; central canals absent. Below spina bifida two halves joined, but cross sections showed two anterior and four posterior horns, with their nerve roots. Central canal here doubled.
(35) Sulzer	Child of few weeks	Lumbar region	Myelo-meningocele. Cartilage mass penetrating obliquely into canal	Doubling, caused by cartilage mass. Between posterior columns a fissure could be traced into 4th ventricle, showing as an open canal in upper sections of cord. Diastematomyelia reached highest point in lumbo-sacral part of medulla. A complete doubling of the cord was found here. (Figures.)

It will be seen from this table that cases 1, 2, 3, 4, and 7 may be put aside as being too imperfectly described to permit of their real nature being determined.

It has been shown by Van Gieson in his valuable article on "The Study of the Artefacts of the Nervous System," in the *New York Medical Journal*, 1892, that the cases of Fürstner and Zacher (14), Kronthal (27), Jakobsohn (30), and Feist (31) are cases of artefacts, an opinion with which we agree. Fürstner and Zacher's case showed cord-deformities of the heterotopic order, with, in addition, a doubling of the cord. These deformities, however, seem to have been due to an injury at the autopsy, and one which was not only sufficient to produce minor displacement of cord substance, but violent enough, at one place, to telescope one portion of the cord down over another so as to double it over a limited space.

Kronthal describes a bruise in the cord of an ox as a congenital malformation. The cord came from a butcher's shop, and was at one point, about 1 cm. long, more voluminous than normal. He remarks that the rest of the cord was "badly

damaged by being sawn in two lengthwise," but it does not seem to have occurred to him that the "anomaly" which he described was also produced by instruments. As in the previous case, the figures illustrating the condition are, we think, quite sufficient proof of the artificial nature of the injury.

Feist describes with thoroughness and detail the topographical and structural changes in the cord due to bruising, but considers them pre-formed and as anomalies in the course of the fibres of the white matter with partial doubling of the cord. The drawings give an excellent idea of how extraordinarily various these artificial doublings are. There is an irregular, fragmentary arrangement of grey and white matter, due to the fusing of different portions of the cord from consecutive levels.

Jakobsohn's case is a bruised cord from an acute myelitis, in which distortions are described minutely and supposed to be congenital malformations. When a cord has been so thoroughly knocked to pieces as his illustrations represent, it is difficult to understand how it can be considered as a malformation, and the case presented as one of heterotopia.

These, therefore, may be also excluded from the list, along with that of Steffen (29), which appears also to have been merely a division produced by injury. Turner's case (33) (in a rabbit) is probably, according to Van Gieson, also an artefact, but it is too shortly described to enable us to form a definite opinion. In any case, both it and Öllacher's (12) specimen of splitting of the medullary tube in a chick embryo may be omitted from consideration here.

Brasch's case (32) is of a markedly degenerate syphilitic cord and seems to us to be of the nature of an artefact or a heterotopia, and not in any sense to be a real doubling of the cord.

When the cases above noted have been eliminated from Steiner's list there still remain 22 of his cases to be considered, and of these 12 (Nos. 5, 6, 10, 16, 17, 18, 19, 20, 21, 22, 25, 26) are examples of mere splitting (*Zweitheilung*—*Diastematomyelia*) of the cord into two parts. All are associated with greater or less degrees of *spina bifida*, and in none of these is there any evidence of real duplication of the cord.

This leaves only 10 cases of doubling (*Doppelbildung*—true *Diplomyelia*) of the spinal cord of man—Cruveilhier (8), v. Ammon (9), v. Lenhossék (11), Foà (13), Recklinghausen (15),

Bonome (23), Miura (24), Rosenberg (28), Chiari (34), and Sulzer (35), to which may be added Steiner's own case, that of Theodor, one reported by von Monakow, and the two cases of the authors, making 15 in all.

Steiner's case was in a child of  $3\frac{1}{2}$  months with a sacral spina bifida and meningocele, but otherwise healthy, and with its functions normal. The central canal was dilated above the level of the division and surrounded by a thickened capsule, much as in our first case. The cord was divided from about the middle of S 1 downwards. In S 2 each cord showed a complete H-form of grey matter, with nerve roots connected with all eight horns.

Theodor's case was a child a week old, with lumbo-sacral spina bifida and meningocele. The doubling was present throughout the lumbo-sacral cord and commenced, as in our second case, with a posterior wedge of grey matter, and the two cords were almost completely formed above the level of their separation. The cords united, separated again, but at the lower extremity they again became united, but with two central canals, four anterior and four posterior cornua. The nerve roots came mainly from the outer halves, but the inner, more dorsally situated cornua had also both anterior and posterior root connections.

V. Monakow's case was that of a cyclopic foetus in which the upper part of the cord was turned back into the skull and formed the roof of the fourth ventricle, splitting the cerebellum. Turning down again it entered the spinal cavity and was doubled in the greater part of its lower course. Higher up one cord became thinner, transformed itself into one half of a cord, and blended with the other into a single cord.

Van Gieson, in referring to Chiari's case, says: "the absence of drawings and definite details of the deformity renders it impossible to draw any conclusion as to the real nature of the changes. . . . (The case would seem to me to look more like a bruise than a malformation.)" We are, however, inclined from the description to think that this is probably a real case of doubling, although not denying the possibility of the condition having been produced accidentally, and we therefore class it along with the cases of Cruveilhier, v. Ammon, Lenhossék, Rosenberg, and v. Monakow as examples of probable true doubling of the cord, of which, however, from the descriptions available, it is

impossible to be absolutely certain. (In the case of v. Monakow we have not been able to see the original full account.) We have, therefore, nine undoubted examples and six probable ones.

Of these we find that 7—the cases of Cruveilhier (8), v. Ammon (9), Rosenberg (28), Chiari (34), Sulzer (35), Steiner, and Theodor were related to spina bifida; that in Cruveilhier's case a cartilaginous projection from the body of the twelfth dorsal vertebra, and that in Sulzer's a similar projection from the fourth lumbar might have produced a backward pressure on the medullary tube in an early stage of its formation, and thus caused a split at a time when the cells were little differentiated, and each half was capable of forming a more or less perfect medullary tube.

In two instances, that of Bonome (23) and our first case, the division has been brought about (or is accompanied) by intrusion of a fold of membrane between the two parts. In our first case a V-shaped fold of dura projected in from the front backwards between the two cords, and some single tags joined this fold with the posterior layer of the dura.

In Bonome's case the cord was split by a layer of connective tissue which contained a small piece of cartilage.

The cases of v. Lenhossek and v. Monakow were in foetuses, and in them and in the cases of Foà, Recklinghausen, Miura, and ourselves (2) the bony canal seemed to be quite normal. In the five last cases the ages of the patients were (Foà) 76, (Recklinghausen) 31, (Miura) 53, (ours) 31, and 50.

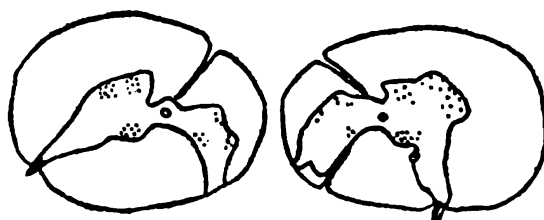
The tracings on the following page, taken from illustrations of the cases accessible to us, show that whether the separation of the two cords has been complete or not, the outer cornua are in every instance the more perfect; the inner, more dorsal cornua being less well formed. The constancy of this feature, the regularity of the arrangement of the superadded cornua, and the fact that in all cases the continuity of the central canals could be traced, all point to the double cord not being of the nature of a foetal inclusion. The only satisfactory explanation seems to lie in a local doubling of the medullary tube. Once this has been formed it becomes intelligible why the inner cornua are not so perfectly formed as the outer, which would still represent the outer halves of the normal tube. But as regards the causation of the doubling, it must be admitted that we can say very little.



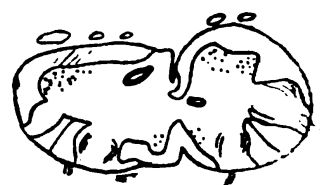
THEODOR.



V. RECKLINGHAUSEN.



SULZER.



STEINER.



MIURA.

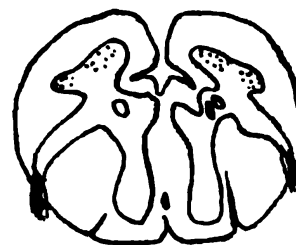






FIG. 1.



FIG. 2.

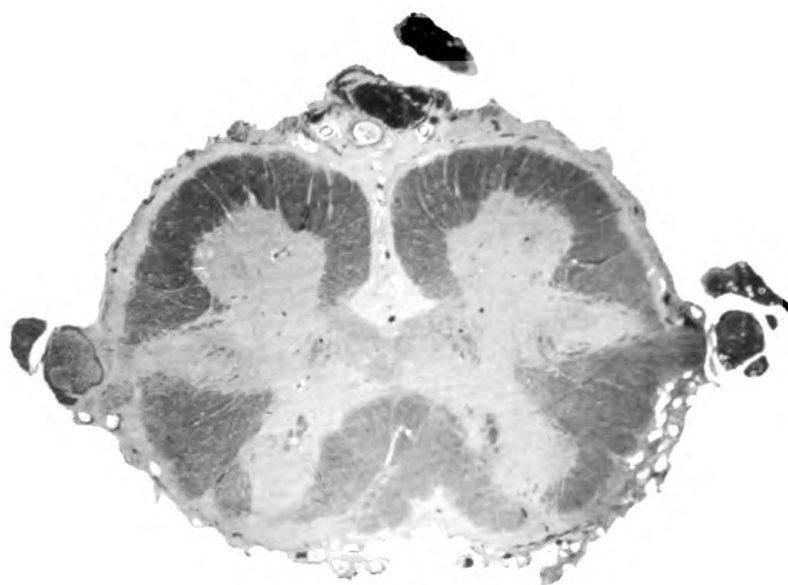


FIG. 3.



FIG. 4.

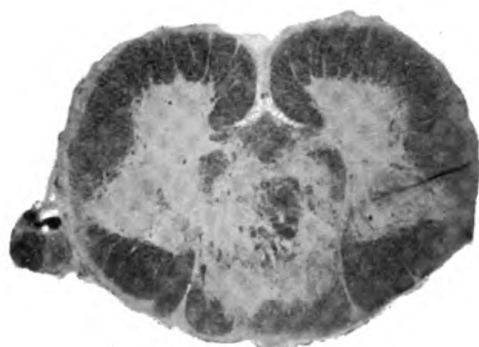


FIG. 5.

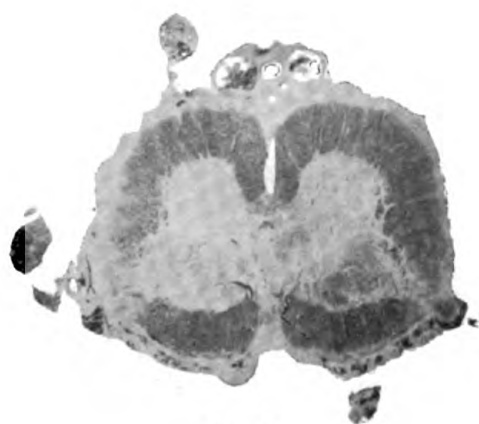


FIG. 6.



The simplest explanation would be to suppose pressure acting either in front or from behind, and in certain cases (Cruveilhier, Sulzer, Bonome, and our first case) there are signs of pressure having acted from the anterior surface. Other modes of doubling of the medullary tubes, such as kinking, can be conceived, but there is no evidence of this having been the cause in any of the cases described.

Finally, in some instances no external cause is evident; the abnormal development may have been from some inherent defect in the cells of the medullary tube itself at a very early period in development, and the doubling affected only the spinal cord, without exerting any influence on the surrounding meso-blastic structures.

#### DESCRIPTION OF FIGURES.

FIG. 1 ( $\times 6$ ) S 1 upper.—Shows the central canal elongated antero-posteriorly and lying in a narrow septum of grey matter.

FIG. 2 ( $\times 6$ ) S 1 lower.—Two central canals. Wide wedge of grey matter intruded between the posterior columns; the hinder part shows a double substantia gelatinosa.

FIG. 3 ( $\times 6$ ) S 2.—Shows normal outer anterior and posterior cornua with nerve roots in connection. The inner, more dorsal halves have well formed posterior cornua; more rudimentary anterior cornua, in contact behind the anterior median fissure. Two distinct posterior "median" septa. A fissure partly divides the cords where they are united posteriorly.

FIG. 4 ( $\times 6$ ) S 3.—Well marked  $\Lambda$ -shaped anterior median fissure. Two small posterior median septa on the outer side of either inner posterior cornu. A fissure partly dividing the cords in the middle line posteriorly. Inner anterior cornua almost separated; the left contains a few motor cells, from which fibres can be seen passing out by the anterior median fissure.

FIG. 5 ( $\times 6$ ) S 4 upper.—Outer anterior and posterior cornua distinct; inner anterior cornua practically disappeared; posterior crushed together and their substantiæ gelatinosæ united. Anterior part of the posterior columns has been cut off as an islet of white fibres.

FIG. 6 ( $\times 6$ ) S 4 lower.—Single central canal. Merely a small band of grey matter in the line of the posterior median septum, representing the fused inner posterior cornua.

## ERRATA.—A Case of Localised Doubling of the Spinal Cord.

This Review, November 1905.

Plate 26, Fig. 11, should be Fig. 10, and is upside down.

Plate 27, Fig. 3, should be Fig. 4.

Plate 27, Fig. 4, should be Fig. 3.

Plate 28, Fig. 7, should be Fig. 8.

Plate 28, Fig. 8, should be Fig. 7.

Plate 29, Fig. 10, should be Fig. 11.

## LITERATURE.

Steiner. "Über Verdoppelung des Rückenmarks," *Inaug. Dissert.*, Königsberg, 1895.

Theodor. "Ein Fall von Spina bifida mit Doppelteilung des Rückenmarks," *Arch. für Kinderheilk.*, Bd. xxiv., 1898.

v. Monakow. "Verhandlung der Naturforscherversammlung," 1896; *Ref. Neurol. Centralbl.*, 1896.

Bruce, M'Donald, and Pirie. This Review, Nov. 1905.

Steiner gives the following list of references to the literature :—

v. Recklinghausen. "Untersuchungen über Spina bifida," etc., *Virchow's Arch.*, Bd. 105, 1886.

C. P. Ollivier. "Traité des maladies de la moelle épinière, Paris, 1837.

Zacchias. "Quæstiones medic. leg."

Manget. "Theat. anatom."

Hull. *Mem. of the Soc. of Manchester.*

Sandifort. "Museum anatomicum academæ Lugdun," Batavæ.

Natorp. "De spina bifida," *Dissert.*, Berlin, 1838.

Cruveilhier. "Anatomie patholog."

v. Ammon. "Die angeborenen chirurg. Krankheiten des Menschen."

v. Lenhossék. "Über eine Zwillingsbildung der Medulla spinalis," *Wochenblatt der Zeitschrift der Wiener Ärzte*, 1858, No. 52.

J. Ollacher. "Über einen Fall von partieller Multiplicität des Rückenmarks in einem 4-tägigen Embryo," *Bericht des naturwissenschaftlichen medizinischen Vereins zu Innsbruck*, Bd. iv., 1875.

P. Foà. *Rivist. speriment. di Freniatria e Medic. legale*, 1878.

Fürstner u. Zacher. "Über eine eigentümliche Bildungsanomalie des Hirns und Rückenmarks," *Arch. f. Psychiat.*, Bd. xii., 1882.

Van Gieson. *New York Medical Journal*, 1892.

E. D. Bondurant. "Duplication of the spinal cord as a result of post-mortem injury," *The Medical News*, 1894.

R. Benecke. "Ein Fall von unsymmetrischer Diastematomyelie, Festschrift für E. Wagner von seinen Schülern," Leipzig, 1888.

A. Bonome. *Archive per le scienze med.*, "Di un caso raro di sdoppiamento parziale del midollo spinale," *Referat. Neurol. Centralbl.*, vii., 1888.

Miura. "Zur Genese der Höhlen im Rückenmark," *Virchow's Arch.*, Bd. 117, 1889.

D. Fischer. "Über die lumbo-dorsale Rachischisis mit Knickung der Wirbelsäule," *Ziegler's Beiträge*, Bd. v., 1889.

P. Kronthal. "Zwei patholog. anatom. merkwürdige Befunde an Rückenmark," *Neurol. Centralbl.*, 1890.

Siegfried Rosenberg. "Über Spina bifida und Diastematomyelie," *Dissert.*, Freiburg, 1890.

Steffen. "Spina bifida, Zweiteilung des Rückenmarks. Hydromyelia," *Jahrbuch für Kinderheilkunde*, 1890.

Louis Jakobsohn. "Ein Fall von partieller Doppelbildung und Heterotopie des Rückenmarks," *Neurol. Centralbl.*, x., 1891.

Bernhard Feist. "Ein Fall von Faserverlaufsanomalieen und partieller Doppelbildung im Rückenmark eines Paralytikers," *Neurolog. Centralbl.*, x., 1891.

Martin Brasch. "Ein unter dem Bilde der tabischen Paralyse verlaufender Fall von Syphilis des Centralnervensystems," *Neurol. Centralbl.*, x., 1891.

Aldren Turner. *Brit. Med. Journ.*, 1891.

H. Chiari. "Über Veränderungen des Kleinhirns infolge von Hydrocephalie des Grosshirns," *Deutsche med. Wochenschrift*, 1891, No. 42.

Paul Sulzer. "Ein Fall von Spina bifida verbunden mit Zweiteilung und Verdoppelung des Rückenmarks," *Ziegler's Beiträge*, Bd. xii., 1893.

### PARAMYOCLONUS EPILEPTICUS.

By ERNEST S. REYNOLDS, M.D. (Lond.), F.R.C.P.,

Senior Assistant-Physician to the Manchester Royal Infirmary,

Physician to the Manchester Workhouse Infirmary.

CASES of paramyoclonus epilepticus are so rare that the following may be found of some interest :—

Benjamin B. C., aged 44, a plumber, was sent to me on Oct. 9, 1905, by my friend Dr Alan M'Dougall, the Medical Superintendent of the David Lewis Epileptic Colony, Great Warford, Cheshire, and was admitted the same day at the Manchester Royal Infirmary under my care. Dr M'Dougall wrote : "He has a daily seizure lasting on an average four hours. His muscles contract in a way that suggests applications of the battery. Every subcutaneous muscle seems to be affected by the spasms. During the attack the patient is unconscious, but the coma does not seem to be absolute. He has had seizures for several years. Occasionally he misses a day, more frequently he has two on the same day. The attack is during the daytime, usually about the middle of the day."

*History of Illness.*—The patient complains of having "fits,"



which commenced with slight seizures while at his work some eight years ago, and they have increased in intensity and frequency since. He cannot suggest anything as a cause of the attacks.

*Family History.*—His father died at the age of 64 and his mother aged 54, causes of death unknown. He is one of 13 children, 10 of whom are still alive and well. He is married and has had 7 children, 4 of whom are living. There is no history of "fits" or other nervous affection in the family.

*Personal History.*—The patient has occasionally suffered from bronchitis, but apart from this has been healthy. He has never had syphilis or lead poisoning (he has no blue line on the gums). He has never taken alcohol in excess; he does not smoke.

#### PHYSICAL SIGNS AND SYMPTOMS.

If no fit is occurring, the patient lies in bed apparently quietly (except for occasional sudden shock-like movements) and talks rationally; he is indeed very intelligent. He is a well-built man, 5 ft. 10 in. in height, and weighs 14 stone 3 lbs.; his muscular system is particularly well developed. His face shows in all parts, especially on the forehead, strongly marked folds, very similar to the "rugosities" of the face seen in an old-standing case of spastic diplegia of infancy. I am of opinion that these folds and the general great muscular development are largely due to the excessive muscular action which is constantly manifested.

He can get out of bed and walk in a perfectly normal manner, except of course when a fit is occurring. But if, while he is lying in bed without his attention being specially diverted, he is very closely observed, it is noticed that there occur from time to time, at irregular intervals varying from a few seconds to perhaps half a minute, sudden shock-like contractions (similar to single contractions produced by single shocks of an induced current) of various muscles or parts of muscles in entirely different parts of the body, either the face, neck, arms, legs, or trunk. Naturally if the whole of a muscle is affected, a bodily movement occurs, but if only a part of a muscle, practically no movement results. So uncertain is it which muscle will contract after any other, that it is necessary to examine the patient

entirely stripped or it might seem for many minutes that no contractions were occurring. An easy method of noticing these shock-like contractions is to place the hand on the patient's forearm or round his leg or resting on his thigh, when, after a short interval, the contractions, however slight, will be felt. The contractions occur on both sides, but not necessarily symmetrically or synchronously. If the patient is engaged in light conversation the contractions may still go on, but if his attention is *strongly* attracted they are perhaps lessened in frequency. There are no contractions of the muscles moving the eyeballs, but contractions of the tongue occur (especially during the "fits"). The movements are present during sleep.

*The "fit."*—In addition to the above movements which occur during complete consciousness, the patient has a "fit" practically every day, commencing about 10 A.M. and lasting about two hours. Sometimes he has a second "fit" in the evening. The attack commences with a complaint of headache and drowsiness, and the patient is then seen to be scratching or rubbing the right side of the head in a clumsy way, generally with the front of the right wrist, the hand being hyperextended. Then rapid irregular clonic contractions of the right fingers and hand occur, the thumb usually being affected first. This is soon followed by clonic contractions of all the muscles of the body, each contraction being sudden and shock-like in character. If watched for a considerable time the contractions may be seen to affect symmetrical muscles on the two sides, but not synchronously; but in the face, neck, and trunk they may be synchronous. It will be noticed that these sudden movements are similar, but much more frequent and more marked, to the movements described above as occurring in the interparoxysmal period. But during the whole time of the fit there are, in addition, greater and relatively slower movements; these are movements of contortion very similar to the choreiform movements of an old-standing case of spastic diplegia, and they affect all parts of the body, face, neck, trunk, arms, and legs, the movements being so marked that the body and limbs are thrown about in all directions and the bed-clothes tossed hither and thither; but the patient never falls out of bed, nor does he bruise himself to any great extent against surrounding objects, although he has knocked the paint and broken the plaster of the wall next his bed (which is in a

corner of the ward) in a patch about 4 inches square. His face is thrown into marked contortions, his lips and tongue being also involved, but he does not bite his tongue. He does not cry out either at the commencement of, or during a fit, but all the time grunts and "snorts" in a peculiar manner, these noises being apparently due to irregular movements of the muscles of respiration and vocalisation; there are no sudden ejaculations of any kind. At first sight all these large irregular movements and contortions resemble closely the "grand movements" of the typical hystero-epilepsy of Charcot, but they are certainly not of this nature, for they are so largely intermingled with the clonic contractions described above. Watching the hand and arm, for instance, the fingers are sometimes extended suddenly, then suddenly flexed; sometimes one or two fingers extended and at the same time the others flexed; whilst at the same time the whole hand is moved about in various directions by the larger movements which affect the whole of the upper limb, these movements being more irregular and slower than the clonic contractions. At the same time (still watching the arm) quick clonic movements may be seen in individual muscles or parts of muscles of the upper arm. And similar appearances showing large movements and simultaneous quick clonic contractions are seen in legs, face, neck, and trunk.

During the fit the patient is *apparently* only partially unconscious. The large irregular movements will occasionally alter if resisted by the examiner, and he apparently actively resents such interference. Sometimes also a sharp loud word of command will cause some alteration; but in a few seconds the movements continue as before. On some occasions if I say to him, "How are you?" he will open his eyes, look towards me, roll his head about awkwardly, and, after an obvious effort, splutter out the words, "How do you do?" and then recommence his movements. But as a matter of fact, from close cross-questioning afterwards, it is quite certain that he is entirely unconscious during the whole period of the attack.

The fit terminates very gradually, as it began; the movements gradually lessen and consciousness gradually returns. There is no incontinence of urine during the fit. No typical post-epileptic sleep follows the attack.

*True Epileptic Attacks.*—During his residence of eight months

at the Epileptic Colony, Dr M'Dougall informs me that the patient had two genuine epileptic fits. While at the Royal Infirmary he had a true epileptic fit lasting a few minutes on November 4, in which he bit his tongue, and another short attack on November 8.

*General Nervous System.*—As I have already stated, the muscular system is everywhere well developed, and there is of course no paralysis. There is nowhere any affection of sensation. The knee-jerks and tendo Achilles-jerks are present, normal and equal on the two sides. The plantar reflexes are of the flexor type; the general superficial reflexes are everywhere normal. The patient says his memory is gradually failing, but there is no evidence of any other mental deterioration.

All other bodily organs are healthy and normal. Urine normal.

#### COMMENTARY.

Because of the apparently irregular large movements of the body and limbs and the peculiar contortions of the face, and the resistance by the patient to any interference during the attack, and especially because the unconsciousness did not seem complete, several observers who saw this case declared, and persisted for some time in believing, that the man was a malingerer. But the finer clonic shock-like muscular contractions, both during the fits and in the intervals, quite dispose of this view, and especially so as some of the clonic spasms only affected parts of a muscle, a condition which could not by any possibility be simulated.

Similarly the possibility of hysteria may be excluded, not only on account of the clonic spasms in isolated muscles or parts of muscles, but because of the occasional occurrence of true epileptic attacks, and also because of the entire absence of the usual hysterical stigmata.

Although, as I have noted above, the large irregular movements were somewhat similar to the "grand movements" occurring in the hystero-epilepsy as described by Charcot, yet the absence of the other typical stages of such attacks and the smaller clonic spasms of the inter-paroxysmal period sufficiently distinguish the condition.

These small clonic shock-like contractions similarly exclude any form of chorea, either Sydenham's, Huntington's, or senile;

and the unconsciousness during the "fits," as well as the genuine epileptic seizures, enable one to say that this is neither a case of electric chorea nor of convulsive tic.

*Pathology.*—The history of this case, taken together with the impressions conveyed to my mind in watching the movements, seem to me to be of some little service in determining the seat of origin of this strange and uncommon affection.

The true epileptic fits which have occurred on at least four occasions during the last twelve months must, I think, be ascribed to some affection of the cerebral cortex. The ordinary daily "fit" must also be due to some affection of the higher or lower cerebral centres, for it is accompanied by loss of consciousness (certainly not always of the most profound type), and by the large, irregular, comparatively slow movements so closely resembling chorea or the choreiform movements of spastic diplegia that the situation of the lesions must almost necessarily be in about the same regions.

But to my mind the smaller clonic shock-like movements, which occur at all times at irregular intervals, can only be due to some affection of the lower motor neurones of the pons, medulla, and spinal cord. The sudden contractions of isolated muscles or even parts of a single muscle, one cannot conceive to be of cerebral origin. They occur, as I have said, at all times: during sleep, during the daily "fit" (when they are much worse), or between the "fits." The increase of the clonic movements during the "fits," when cerebral control is cut off, is also easily explained. They occur also without consciousness being necessarily lost.

In this case, then, the disease seems to be due to an affection, possibly some slow degeneration, of higher and lower cerebral centres, and also of the motor centres of the pons, medulla, and spinal cord.

---

# **LESIONS OF THE SPINAL CORD, THE RESULT OF ABSORPTION FROM LOCALISED SEPTIC FOCI, WITH A PRELIMINARY NOTE ON AN EXPERIMENTAL RESEARCH.<sup>1</sup>**

By DAVID ORR, M.D., and R. G. ROWS, M.D.

IN a paper published in the Winter number of *Brain*, 1904, while discussing the starting-point and distribution of posterior column lesions in General Paralysis of the Insane, we referred to the work which has been done to determine the course of the lymph stream in the posterior roots and columns, and we stated that it has been proved by several observers that the lymph flows in an ascending direction towards the cord.

A reference to this paper (1) will show the reasons for adopting this view. The object of our present communication is to bring forward further definite evidence in favour of the theory that lesions of the spinal cord can be produced by the ascent of toxins, or in some cases even of organisms, from peripheral foci of inflammation, either of an acute or a chronic nature.

The series, from which our conclusions have been drawn, comprises in all eight cases, which are mentioned in the table below. The lesions were of varying nature and situated in different parts of the body, and in each case, as a reference to the table will show, the cord lesion was found to be much more intense in the segments of the cord corresponding to the nerve supply of the affected area.

---

**CASE 1.** Bedsores ; gluteal region ; more severe on left side ; sup-  
puration of right elbow.

---

Degeneration of the posterior columns and of the anterior radicular fibres in the lumbo-sacral region from S 1 to L 3 ; more intense on the left side and in 4th lumbar. From D 12 to D 2 there was nothing worthy of note. Degeneration again commenced at D 1 on the right side ; the lesion was most marked in C 7 and 6, and gradually diminished in the next few segments.

---

<sup>1</sup> An account of this research will form the subject of a separate paper. Towards the expenses of this research we have received a grant from the British Medical Association.

<b>CASE 2.</b> Left brachial neuritis; staphylococci in the tissues around the posterior root ganglia.	Marked degeneration of the left root-entry zone and Burdach's column from C 8 to C 2; maximum in C 7-6; faded gradually from C 6 upwards. Degeneration also present in the lateral region and anterior radicular zones. Right half of cord affected similarly, but to a markedly less extent. There were no changes in the anterior or posterior roots.
<b>CASE 3.</b> Bedsores on buttocks and sacrum; of longer duration on the right side.	Lesion most intense on the right side. Some degeneration of posterior columns in S 1; maximum at the level of L 4-3; diminished gradually in the segments above this.
<b>CASE 4.</b> Pelvic cellulitis; renal abscesses; double empyema, of longer duration on the left side; abscess in the cervical muscles; no peritonitis.	Very slight degeneration of the posterior columns in the sacral region. Much Marchi reaction amongst the anterior radicular fibres of S 4-3-2, especially on the left side. No changes in the lumbar cord. Degeneration in the root-entry zones of D 11-10-9; again a separate lesion beginning in D 5 occupying the same area, more marked on the left side, and occurring in every segment as high as C 4. In cervical region degeneration greatest in C 7-6.
<b>CASE 5.</b> Caries of the 4th and 5th lumbar vertebræ; psoas abscess on the right side. The abscess cavity became septic two months before death.	In S 2 a slight lesion of the posterior columns. This increased in S 1, and was most intense in L 5-4. Above this level it gradually diminished. Lesion much more marked on the right side.
<b>CASE 6.</b> Chronic suppuration of the right knee-joint.	Degeneration from S 1 to the lower dorsal region; most marked in the right posterior columns and most intense in L 4-3-2.
<b>CASE 7.</b> Chronic suppuration of the left knee-joint.	Lesion of the posterior columns slight in S 1; well marked in L 5-4-3. It ceased at D 11; most intense on the left side. There were no changes in the left sciatic nerve.
<b>CASE 8.</b> Prostatic disease; chronic cystitis.	Followed by transverse myelitis at the level of D 8-9.

An examination of the spinal cords from the above-mentioned cases has enabled us to confirm the two points insisted on before, viz. that system degenerative lesions of the sensory protoneurons always begin at the point where the fibres entering the cord lose their neurilemma sheath, and spread thence into the posterior

columns ; that such lesions in their early stages, and even for a prolonged period, exist without any appreciable changes in the posterior roots.

It is now generally admitted that the posterior column lesions in early Tabes, and in other conditions, such as Diabetes and General Paralysis of the Insane, are primary, and not dependent upon any lesion of the posterior roots, or of the posterior root ganglion cells.

But besides defining the starting-point of these degenerations, our series of cases seems to show clearly, by the distribution of the lesions in the cord, that the changes are the direct result of absorption from some peripheral septic focus. For example, in Case 1, with bed-sores, which were more severe on the left side, the lumbo-sacral enlargement showed considerable degeneration, whereas the dorsal region was almost entirely free. In Fig. 1, note the greater degree of degeneration on the left side ; in Fig. 2 only the centre of the posterior columns shows a few scattered fibres. Further, in the same case, corresponding to a suppuration of the right elbow-joint, there was a marked degeneration of the cervical enlargement, which was more intense on the right side (Fig. 3). Case 2 exhibited the same localised and limited distribution, but on the left side (Fig. 5).

There is evidence, however, that, although the toxins are carried along the perineural sheath into the corresponding posterior column, a certain quantity of the toxic lymph spreads along the loose meshes of the pia mater to the opposite posterior column, and also in a lateral direction to the adjacent portions of the cord (Figs. 3 and 5).

Further, we have found that toxins are carried along the perineural sheath of the motor roots as well, but the resulting degeneration is never so intense as in the posterior columns.

Homen's<sup>1</sup> observation that toxins seem to reach the spinal cord more readily by the posterior than the anterior roots is interesting in this connection. A possible explanation may be found in the fact that the posterior roots are nearly three times as large as the anterior, and can therefore pour a larger quantity of lymph into the cord in a given time.

The degeneration amongst the anterior radicular fibres, as in the case of the posterior roots, commences at the cord margin

<sup>1</sup> Referred to *Brain*, Winter 1904.



where the neurilemma sheath is lost, and affects only the intramedullary portion. We therefore consider this point just as vulnerable to the action of toxins ascending in the perineural lymph stream as the corresponding one on the sensory proto-neuron system. From the point where the anterior roots enter the cord, some of the lymph, instead of passing along the radicular fibres towards and into the grey matter, diffuses laterally, and produces a degeneration of the fibres in the adjacent regions.

There is one other fact to be mentioned, and that is, that in all our cases we have constantly observed degeneration amongst the fibres of the anterior commissure, but only in those segments which exhibit the lesions described above.

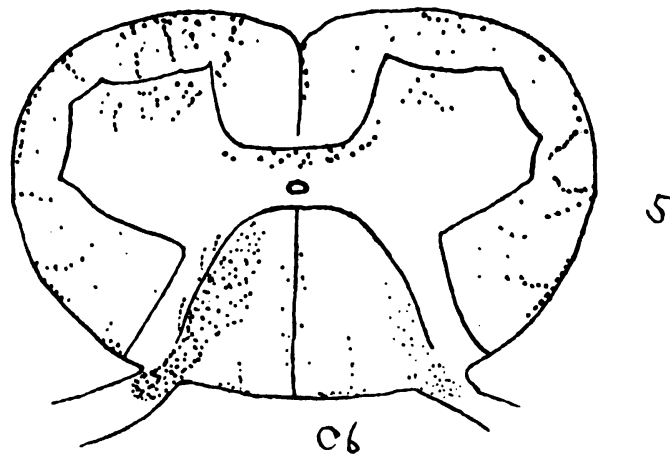
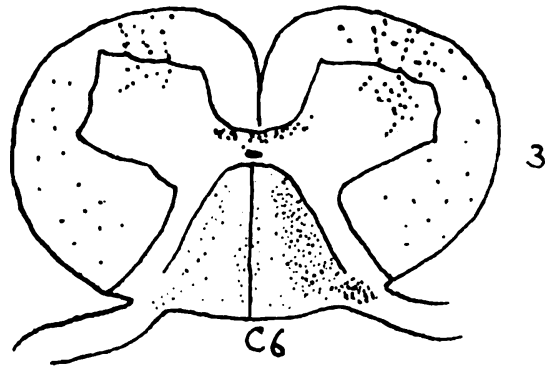
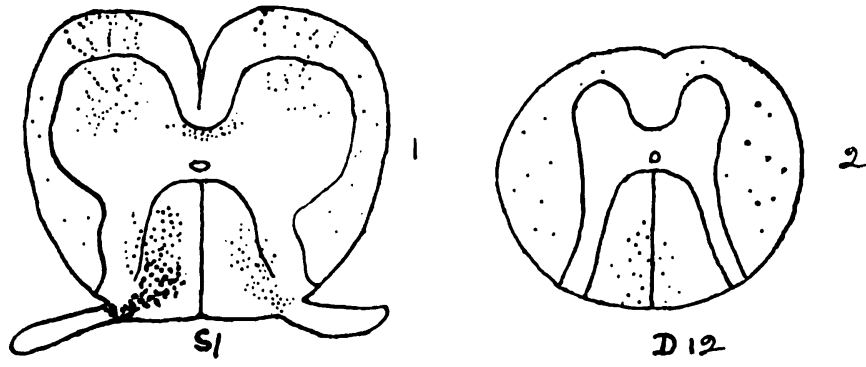
The effect of the lateral diffusion of the toxic lymph referred to above is best seen in Case 2, Fig. 5. From this case—brachial neuritis of infective origin—it seems highly probable that this diffusion from the two points of maximum intensity occurs more readily when the toxins are present in greater quantities, or when they possess a higher grade of virulence. We have failed to find evidence of it in milder degrees of toxicity, such as are associated with bedsores.

Fig. 5 shows the distribution of the lesion in Case 2 as it is demonstrated by the Marchi method.

The degeneration of the posterior columns began at the point where the fibres lose their neurilemma sheath, and spread forwards into the root-entry zone. Amongst the anterior radicular fibres also there was much reaction, which extended from the cord margin, where these fibres also lose their neurilemma sheath. In addition, the diagram shows considerable degeneration around the margin of the cord and along the pial prolongations dipping into the lateral tracts.

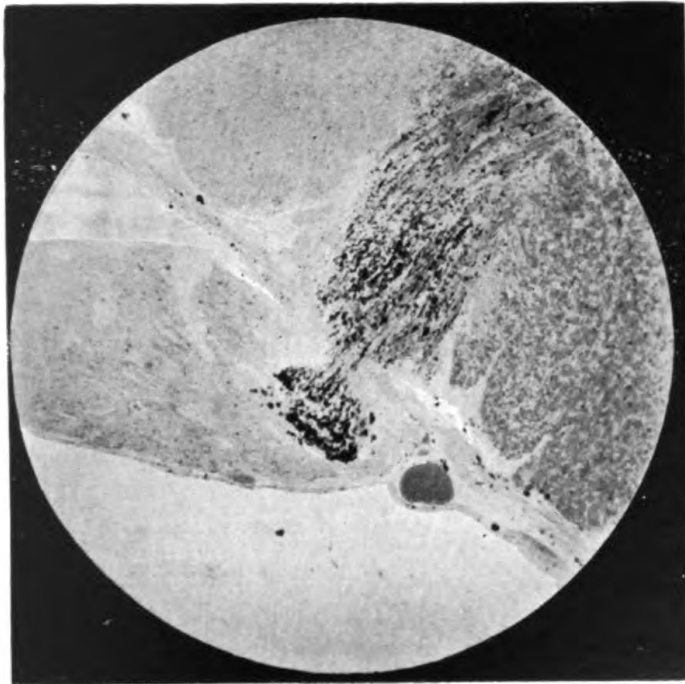
The degenerations in the anterior part of the cord, although present elsewhere, have been most clearly seen and followed even to the cell groups of the grey matter, in the upper sacral and lumbar regions.

After running up to the cell groups, the degenerated fibres encircle them, and change their position with them (Fig. 4). For example, in the sacral region, where the motor cells occupy a postero-lateral position, the degenerated fibres travel through almost the whole depth of the anterior cornu to reach them ;



To show the distribution of Marchi reaction.







while at a higher level, where the cells lie more anteriorly, the Marchi reaction is chiefly confined to this region.

It will thus be seen that there are two paths by which the lymph enters the cord from the periphery, and two points especially open to attack. It is at these two points that we find the maximum amount of degeneration of the nerve fibres, while there is a less severe affection of the fibres in the adjacent regions.

We have recently examined the cord of a case of myelitis, which occurred in the course of a septic cystitis. The myelitic focus was situated in the 8th and 9th dorsal segments, and the distribution of the maximum degeneration suggested strongly to us that the myelitis was the result of absorption from the bladder. We found on examination by the Marchi method that the posterior columns showed most change, while the postero-lateral region was affected to a somewhat less extent. In each anterior radicular zone there was a well-defined patch of degeneration. The more lateral region and the grey matter exhibited only a limited and scattered lesion.

This case, while it differed ætiologically from the others of our series, is highly suggestive of an organismal infection from the bladder, because of the distribution of the myelitic patches in the entry zones of the anterior and posterior roots.

Similar cases are recorded by Walker in the *Lancet* for March 11, 1905. He described three cases in which an acute ascending paralysis occurred in the course of chronic cystitis, and suggested as an explanation that the lesion of the cord was due to an extension of an inflammation along the nerves from the bladder to the cord. Now, all our observations tend to show that from any septic focus, wherever it may be situated, toxins, and in some cases organisms, can ascend to the cord, and there, for the first time, exert their noxious influence. We do not think it probable that the resulting lesion of the cord is due to a direct extension of the inflammatory process, because it is contrary to our experience to find the peripheral nerves or the spinal roots affected.

We therefore still adhere to the view which we have expressed before, that the toxins spread upwards in the perineural sheath without producing any reaction before the cord is reached. With the view of testing the validity of the opinions which we have enunciated in this paper, we have undertaken an experimental

research on animals. By means of these experiments we are able to provide a constant limited supply of toxins in the neighbourhood of some peripheral nerves or spinal roots. So far, the results which we have obtained have shown that the reaction produced in the spinal cord is not a continuation of changes in the nerves, and also that the lesion in the spinal cord commences at the two vulnerable points which we have dealt with above.

In conclusion, we have to thank Professor Lorraine Smith for many suggestions in connection with our work, and for his kindness in allowing us to carry on the experimental part of our research in his laboratory at Owen's College, Manchester.

#### REFERENCES.

1. Orr and Rows. *Brain*, Winter, 1904.
2. Walker. *Lancet*, March 11, 1905.

---

## Abstracts

### ANATOMY.

#### THE DEVELOPMENT OF THE CRANIAL AND SPINAL NERVES

##### (1) IN THE OCCIPITAL REGION OF THE HUMAN EMBRYO.

G. L. STREETER, *Amer. Journ. of Anat.*, Vol. iv., 1904, No. 1.

THE tenth and eleventh cranial nerves are parts of the same complex, both possessing mixed motor and sensory roots and ganglia derived from the same ganglionic crest, but during the process of development the cephalic end of this complex becomes predominantly sensory and the caudal predominantly motor. This produces the appearance of two separate portions and has led to their being considered as two independent structures, the cephalic being known as the vagus nerve and the caudal portion as the accessory nerve of Willis. The ninth (glosso-pharyngeal) nerve is, however, developed quite independently of this complex. In the earlier stages of development (third to fifth week) the ganglionic crest of the vago-accessory complex is an unsegmented structure which extends to the level of the third to fifth cervical segment of the cord, placed on the lateral surface of the latter between the points of later attachment of the dorsal and ventral roots. As development proceeds it splits up into several segments, the most oral and largest develops further and forms the root ganglion of the vagus (jugular ganglion), the more caudal divisions diminish in size spinalwards and remain rudimentary. They are represented in the adult by the ganglion cells which are to be found in the

roots of the accessory nerve. Having developed from the ganglionic crest they give origin to sensory fibres, but these probably join the vagus trunk, as none such are present in the accessory nerve. The root ganglia of these cranial nerves do not present a segmental arrangement. The trunk ganglia of the ninth and tenth cranial nerves (ganglion petrosum and g. nodosum) are not connected with the root ganglia of the same nerves when they can be first identified. They then lie isolated in the mesoderm immediately under the epidermis and are, in contrast to the root ganglia, segmentally related to the gill arches. The root ganglion of ninth nerve (Ehrenritter's ganglion), like those of the eleventh, remains rudimentary. The ventral roots of the spinal nerves develop earlier than the dorsal, and, similarly, those portions of the cranial nerves which are recognised as motor are differentiated into fibre path earlier than their corresponding sensory elements.

The twelfth nerve in young embryos closely resembles the ventral roots of the adjacent spinal nerves and is segmentally continuous in the same line with them. The occasional presence of Froreip's ganglion suggests that a phylogenetic retrogression has deprived the hypoglossal nerve of the dorsal root it once possessed. The dorsal root of the first cervical nerve is similarly often absent.

GORDON HOLMES.

**ON THE CLAUSTRUM.** (*Au sujet de l'avant-mur.*) TROLARD, *Rev. (2) Neurol.*, Nov. 30, 1905, p. 1068.

HITHERTO only one portion of the claustrum has been generally described, viz. its vertical limb which lies beneath the island of Reil, but there is in addition a horizontal portion which extends beneath the putamen.

The only statements the author offers on the connections of the claustrum with the rest of the brain have been obtained by macroscopical dissection. Some of the fibres which leave it pass into the corona radiata, others reach the third frontal, the ascending frontal, the ascending parietal, and the first parietal convolutions. The anterior fibres appear to join the fasciculus uncinatus, the posterior the fasciculus longitudinalis inferior.

GORDON HOLMES.

**THE INFERIOR LONGITUDINAL BUNDLE AND THE CENTRAL**

(3) **OPTIC BUNDLE.** (*Le faisceau longitudinal inférieur et le faisceau optique central.*) LA SALLE ARCHAMBAULT, *Rev. Neurol.*, Nov. 30, 1905, p. 1053.

THIS paper is devoted to a description of the central optic tract based on the examination of pathological specimens by Weigert's



medullary sheath stain. The only origin of the central optic tract is from the external geniculate body, no fibres come from the thalamus. At first it does not form a compact bundle, as the fibres are separated up by the projection fibres which pass between the basal ganglia and cortex through the region of the retrolenticular segment of the internal capsule. Within the temporal lobe some of these fibres are situated in the external sagittal layer ("inferior longitudinal bundle"), some in the internal sagittal layer (Gratiolet's radiations), but in the occipital lobe they all lie in the external layer and are its only constituent. They terminate in the lips of the calcarine fissure. In the frontal portion of their course they are closely intermingled with various systems of association fibres, which should be carefully distinguished from them.

These observations confirm the work of Flechsig, Probst, Redlich, and others, that the inferior longitudinal bundle is a projection and not an association system, and that it, or part of it, represents the central visual tract. As the older name "optic radiations" (of Gratiolet) has been applied to another system which is not part of the visual apparatus, the new term "central optic bundle" is suggested for the geniculo-occipital fibres.

GORDON HOLMES.

#### **A CONTRIBUTION TO THE HISTOLOGY AND DEVELOP-**

##### **(4) MENTAL HISTORY OF THE CEREBELLUM. K. BERLINER,**

*Arch. f. mikr. An.*, lxvi. 2, 5, 220.

THE author considers that the structures described by Denissenko as "Eosinzellen," and which are found in the inner granular layer of the cerebellum of all vertebrata, are no true cells, but constitute a special nervous mechanism of the molecular layer of the cerebellum.

They consist in agglomerations of large and small acidophil granules between which appropriate methods reveal a network of very minute fibres.

It would appear that the granules and the network fibrils are bound together in the acidophil bodies by some sort of ground substance to form a more or less continuous structure.

Certain of the axis-cylinders coming up from the white matter end by dividing into a very fine network surrounding these agglutinations of fibrils and eosinophil bodies.

It is very probable that the terminations of other neurones end in these eosinophil bodies, and that the latter are in connection with one another by direct fibre tracts.

Neuroglia fibrils take no part in the formation of these bodies. Probably these structures have an important function as an association and "schalt" mechanism.

The maximal development of the cerebellar surface in the latter half of foetal life and the first month of extra-uterine life in the human subject, corresponds with the period of rapid disappearance of the superficial granular layer. It is therefore probable that this layer is an indifferent cell-forming material which possesses the power to form not only nerve cells but also glia cells.

F. GOLLA.

**THE RELATION BETWEEN THE OCCURRENCE OF WHITE**

**(5) RAMI FIBRES AND THE SPINAL ACCESSORY NERVE.**

A. H. ROTH, *Journ. Comp. Neur. and Psychol.*, Nov. 1905, p. 482.

IN both the cat and rat there is a sudden increase in the number of white rami fibres in the ramus communicans of the nerve which immediately succeeds the lowest root of the spinal accessory nerve, *i.e.* considerably above the level of the uppermost *obvious* white ramus. It does not follow that the cervical portion of the spinal accessory nerve actually represents the white rami fibres of the upper cervical spinal nerves; probably the downward extension of the vagus nucleus as spinal accessory nucleus has simply displaced downwards the cell column from which the white rami fibres take their origin. No demonstration, however, is given of the presence of such a column in the middle cervical region.

J. H. HARVEY PIRIE.

**THE WIDTH OF THE CORTEX AS A FACTOR IN THE ESTIMA-**

**(6) TION OF THE DEVELOPMENT OF THE BRAIN AND**

**INTELLIGENCE.** (*Die Rindenbreite als wesentlicher Factor zu Beurtheilung der Entwicklung des Gehirns, etc.*) THEODOR KAES, in Hamburg, *Neurolog. Centralbl.*, Nov. 16, 1905, p. 1025.

THE author has amplified the original observations of Schwalbe, who was the first to adopt as a criterion of the development of the intelligence, in addition to observations on the weight of the brain, estimations of the thickness of the cortex and the relative number of ganglion cells.

Kaes considers that observations of the width of the cortex in relation to the developmental history of the various medullated tracts opens a field which promises great results in the study of cortical localisation by histological methods.

In this paper the observations made on thirty-two brains are represented graphically.

He claims that his results will, when sufficiently amplified, afford a basis for an exact knowledge of the relations between nerve cells and nerve fibres and the development of the intelligence in normal subjects and the psychoses of pathological brains. The special activity of individuals who have devoted themselves to some one branch of thought or muscular exercise should, it is claimed, be manifest in an anatomical analysis such as has been undertaken by the author. F. GOLLA.

**THE CEREBRAL CORTEX OF THE DOLPHIN.** [Il mantello cerebrale del delfino (*Delphinus Delphis*).] V. BIANCHI, *Annali di Neurol.*, Fasc. 6, 1904.

AFTER mentioning the lobes, chief convolutions, and sulci of the cortex of the brain of the dolphin (*Delphinus Delphis*), the author describes the arrangement of the layers of the grey matter of the cortex in different regions of the brain, and of the number and appearance of the nerve cells in these layers.

The most striking feature, macroscopically, is the small proportions of the frontal lobes. Almost the whole mass of the brain is included in the parieto-occipital lobes, the frontal lobes appearing as thin layers in front of these. The temporal lobes are also small, and the olfactory lobes are wanting.

Microscopically, it is found that the number of layers in the cortex, and the number of cells in the layers, varies very much in the different regions. In the frontal lobes the cells are not numerous, they have an irregular triangular shape, and few protoplasmic processes. In the parieto-occipital lobes the cells are more numerous, they are larger, many of them are almost pyramidal in shape, and they have more processes. In the hippocampal region the cells may be pyramidal, fusiform, or triangular.

The author then shows the intimate relation which exists between the neuroglia and the nerve cells; the neuroglial fibres form both a pericellular and an intracellular network. He saw no anastomoses between the terminations of the protoplasmic processes of different nerve cells.

Finally, the author attributes the great stupidity and limited psychic activity of the dolphin to the poor development of the frontal lobes—the principal seat of the associative processes—to the uniformity of the constituent elements of the cerebral cortex and to the rarity of the giant pyramidal cells. R. G. ROWS.

**THE STRUCTURE OF THE SPINAL CORD OF THE OSTRICH.**

(8) G. L. STREETER, *Amer. Journ. of Anat.*, Vol. iii., 1903.

THE spinal cord of the ostrich consists of fifty-one segments, each of which gives origin to a pair of motor (ventral) and receives

a pair of sensory (dorsal) roots. Between the seventeenth and twenty-first segments the cord is slightly enlarged—brachial enlargement, corresponding to the segmental level of the wings; otherwise it is almost uniform in size and appearance, till the twenty-sixth segment. Here the lumbo-sacral enlargement begins and extends to the thirty-seventh segment. Below the latter level the cord decreases abruptly in size. A peculiarity which the cord shares with that of all other birds is a separation of the dorsal columns in the lumbar region—in the ostrich from thirty-first to thirty-sixth segments—so that a large fossa appears between them. This is filled with gelatinous-looking material which on microscopical examination is seen to consist of large vacuolated cells, and which Streeter regards as metamorphosed neuroglia, as similar material surrounds the whole cord at this level except at the attachment of the lateral pial ligament. The embryological evidence quoted by Kölliker is also in favour of this view. The dorsal (grey) commissure is absent within these segments. At the same level the ventral fissure is also widened so as to form a ventral fossa. Owing to the increase of ventral horn cells at the levels of exit of the roots in the lumbo-sacral enlargement, definite projections (*eminentiæ ventrales*) are visible on the ventral surface of the cord.

The only other noteworthy peculiarity in the arrangement of the grey matter of the cord is the existence of isolated masses of cells in the periphery of the ventro-lateral columns, as have been described by Lachi and Kölliker in other birds. Six pairs, containing large nerve cells, are present in the lumbo-sacral enlargement on the lateral margins of the cord, and similar but smaller groups with less well-developed cells are found at the root levels of the brachial enlargement.

The paper contains no definite information on any of the tract systems of the white matter of the cord, but as the dorsal columns do not increase in size cerebralwards, it is assumed that only a small proportion of their fibres reach the higher centres directly.

GORDON HOLMES.

## PHYSIOLOGY.

### A RESPIRATORY CENTRE IN THE CEREBRAL CORTEX OF

- (9) THE DOG, AND THE COURSE OF THE CENTRIFUGAL FIBRES WHICH ARISE FROM IT. (*Ueber ein Athemcentrum in der Grosshirnrinde des Hundes und den Verlauf der von demselben entspringenden centrifugalen Fasern.*) C. MAVRAKIS and S. DONTAS, *Arch. f. Anat. u. Physiol.*, H. 5 u. 6, 1905, p. 473.

In the brain of the dog these authors have located a small area in the upper part of the anterior central convolution, stimulation of

which produces definite changes in the respiratory rhythm, unaccompanied by any other muscular movements. Beyermann has described two cortical centres, one in the posterior and the other in the anterior part of the above-mentioned convolution. Irritation of the former, he stated, brought respiration to a standstill, with the thorax in the position of forced inspiration, while excitation of the latter produced respiratory acceleration. The present authors, from their own experiments, believe that there is only a single centre, and that both these effects can be obtained by varying the strength of the stimulus applied to it—weak interrupted induction shocks leading to acceleration of the respiratory movements, strong shocks bringing these movements to a standstill. If the current was made stronger still, movements of the head were observed, owing to spread into the neighbouring centre for the neck muscles.

Having determined the position of the cortical respiratory centre on each side, they made experiments to find out whether the centrifugal fibres from that to the centres in the medulla oblongata were direct (homolateral) or crossed. After a mid-brain hemisection, stimulation of the cortical centre on the same side produced no effect on the respiratory movements, while stimulation of the centre on the opposite side did. If the incision extended slightly across the middle line, involving the whole of one-half of the mid-brain and the mesial part of the other half, no effect on the respiratory rate was produced by stimulation of either cortical centre. When an incomplete hemisection was made, the incision not extending quite to the middle line, irritation of both cortical centres led to the usual respiratory changes.

The cortico-bulbar respiratory tract, therefore, is entirely homolateral, at least as far down as the level of the mid-brain, and it lies close to the middle line.

SUTHERLAND SIMPSON.

**CENTRAL RESPIRATORY INNERVATION.** (Zur Lehre von der (10) **centralen Atheminnervation.**) R. NIKOLAIDES, *Arch. f. Anat. u. Physiol.*, H. 5 u. 6, 1905, p. 465.

FROM experiments performed on rabbits and dogs the author has arrived at the following conclusions:—

1. In the medulla oblongata is situated a centre which controls the muscles of ordinary respiration, by ordinary respiration being meant active inspiration and passive expiration. In addition to this, the presence of another centre for active expiration must be assumed.

2. The ordinary respiratory centre in the medulla is acted upon by a higher centre situated in the posterior corpora quadri-

gemina, destruction of which produces the same effect on the respiratory movements as division of both vagi. Normally this centre has an inhibitory action on the inspiratory phase of normal inspiration similar to that exercised through the undivided vagi. Removal of this centre has, therefore, the same effect as division of the vagus—a slowing and deepening of the respiratory movements due to an increased prolongation of the inspiratory phase. This effect is evident even with the vagi intact, but it becomes much more so if these nerves are divided also. There are thus two paths through which impulses inhibiting inspiration in ordinary breathing can be transmitted to the above-named centre in the medulla oblongata, and there is reason for believing that one of these can compensate for absence of the other. The result of division of both vagi in three dogs was a reduction of the respiratory rate to about a third of the normal immediately after the operation, but at the end of from twenty-five to thirty-five days it had returned almost to the normal. This return to the normal rate might be explained by supposing that the higher inspiration inhibitory tract coming from the centre in the posterior corpora quadrigemina had vicariously taken on the function of the vagi.

3. Similarly, in the anterior corpora quadrigemina is found a centre which acts upon the expiratory centre in the medulla oblongata, so as to inhibit its activity in normal breathing.

4. In various parts of the brain above the medulla oblongata there have been described, by different authorities, inspiratory centres, *e.g.* between the corpus striatum and optic thalamus, in the floor of the third ventricle, at the junction of the anterior and posterior corpora quadrigemina, etc. These do not exist. The respiratory changes on which these statements are based have probably been brought about by stimulating the inspiratory tract at different levels in its course from the cortical respiratory centres to those in the medulla oblongata. SUTHERLAND SIMPSON.

#### **THE CONTRACTILE MECHANISM OF THE GALL-BLADDER**

(11) **AND ITS EXTRINSIC NERVOUS CONTROL.** F. A. BAINBRIDGE and H. H. DALE, *Journ. of Physiol.*, Nov. 9, 1905, p. 125.

IN a series of experiments on dogs the changes in volume of the gall-bladder were investigated by the introduction into the fundus of the bladder of a catheter bearing on its end a rubber bag which was distended with water at a known pressure. The changes in pressure were recorded by a small Hürthle piston-recorder in communication with the pressure reservoir. Extensive dissections

were necessary to prevent extraneous pressure on the gall-bladder by the abdominal walls, diaphragm, and the liver itself. The abdomen was opened freely, sternum and diaphragm divided, phrenic nerves cut, and the gall-bladder carefully separated from the liver. The animal was immersed in warm salt solution, or protected from cooling by frequent application of flannels soaked in warm saline and placed over the liver.

The gall-bladder shows rhythmical contractions at the rate of from 1-3 per minute. This rhythm is intensified by section of the splanchnic nerves, by injection of chrysotoxin, and occasionally by increase of pressure in the gall-bladder.

Stimulation of the right splanchnic nerve produces inhibition of tone and relaxation of the gall-bladder. Stimulation of the left splanchnic has either no effect or causes a slight increase of tone and rhythm, in all probability the result of the general rise of blood-pressure. The injection of adrenalin into the blood-vessels has a similar effect to stimulation of the right splanchnic. The relaxation of the gall-bladder is followed by a gradual after-increase of tone, for which the improvement of blood-supply is probably responsible. The contraction of the gall-bladder following stimulation of the splanchnics and injection of adrenalin which had previously been noted by other observers is really to be ascribed to the engorgement of the blood-vessels of the liver pressing on the gall-bladder. The right splanchnic nerve, however, contains some motor-fibres, and their effect can be produced when the tone is lowered by enfeeblement or stoppage of the circulation.

Both vagi contain motor-fibres to the gall-bladder, the left being more effective than the right. Stimulation causes increase of tone and of the rhythmic contractions. The effect is abolished by atropin.

Anæmia produced by occlusion of the thoracic aorta decreases the tone. Nicotine, bile-salt, atropin, and amyl nitrite also produce relaxation. Pilocarpine and peptone cause apparent contraction, but this is solely due to swelling of the liver pressing on the gall-bladder.

The experiments do not show any contraction of the gall-bladder following the application of acid or the products of gastric digestion to the duodenal mucosa or the biliary papilla, nor any contraction on electrical stimulation of the biliary papilla, or on rapid distension of the gall-bladder. The authors, however, state that they draw no conclusion from this as to the existence of such reflexes in unanæsthetised animals.

PERCY T. HERRING.

**THE PARALYSIS OF INVOLUNTARY MUSCLE.** Part II. On (12) paralysis of the sphincter of the pupil, with special reference to paradoxical constriction and the functions of the ciliary ganglion. H. K. ANDERSON, *Journ. of Physiol.*, Nov. 9, 1905, p. 125.

THE pupil after excision of the ciliary ganglion or oculo-motor nerve sometimes becomes smaller than the one which has its nerve supply intact. Although the sphincter pupillæ is paralysed, the muscle under certain conditions is contracted. Anderson calls this "paradoxical pupil-constriction," a term corresponding with that of "paradoxical pupil-dilatation," in which there is dilatation of the pupil after section of the cervical sympathetic or excision of the superior cervical ganglion.

Anderson by experiments on cats excluded the possibility of there being any nerve fibres from the sympathetic in the ciliary ganglion, or the possibility of injury to the dilatator fibres in removal of the ganglion or section of the short ciliary branches near their origin.

After excision of the ciliary ganglion the pupil of that side is widely dilated under normal conditions, but if the animal is killed a few days after the operation the pupil soon after death begins to contract, and in some hours' time is tightly contracted, while the other pupil has dilated. The same effect is produced by dyspnoea under an anæsthetic, but not so readily as is the phenomenon of pupil-dilatation after section of the sympathetic; the latter is easier to produce, and comes on sooner. Local paradoxical constriction of the pupil can be brought about by section of individual short ciliary branches close to the ganglion.

The probable cause of paradoxical constriction is a primary increase of excitability of the muscle after paralysis, and some local stimulation of the sphincter. The latter may be the lack of oxygen, excess of carbonic acid, or the formation of some substance such as sarcolactic acid.

In a further series of experiments the oculo-motor nerve was divided in one orbit, and the ciliary ganglion removed from the other. In some a slight inequality of the pupils resulted, but did not persist. There is no evidence of the decentralised ganglion having any power of sending out augmentor or inhibitory impulses.

After removal of the ciliary ganglion no fibres degenerate in the third, fourth, fifth, or sixth nerves, so that the ganglion does not contain cells comparable with those of the posterior root ganglia. Removal of the ganglion does not cause lesions of the cornea.

PERCY T. HERRING.



- THE LAWS OF ERGOGRAPHY, A PHYSIOLOGICAL AND**  
 (13) **MATHEMATICAL INVESTIGATION.** (*Les Lois de l'Ergographie, étude physiologique et mathématique.*) J. IOTYKO,  
*Ann. d'Electrobiol. et de Radiol.*, No. 2, 1905, p. 259.

WE have here the first instalment of what promises to be an interesting and valuable contribution to that growing body of literature in which an attempt is made to introduce the exact methods of mathematics into physiology. In an age which has seen Sir William Ramsay conjure new elements into existence by mathematical abracadabra, and Professor Thomson lay down the law to the very constituents of the atoms, the efficacy of the methods cannot be disputed. The only doubt is whether our knowledge of physiological processes is sufficiently detailed to allow of their application. In the introductory part of her paper, Mlle. Ioteyko deals with this question with special reference to ergograms.

While admitting that our knowledge of the course and result of movement is not sufficiently intimate for us to predict its law *a priori*, the writer nevertheless believes that we may be able to discover empirically a formula which shall not only serve as a concise expression of our present knowledge, but also as an instrument to guide further research.

Every ergogram is a curve, and as such lends itself to mathematical expression. The equation to a curve is simply such an expression of the relation between its constituent parts as enables us to draw the curve. To take a simple example. The curve of fatigue described by the calf muscle of a frog has been shown by Kronecker to be a straight line: that is, each contraction differs from the one which precedes it by the same amount. The equation to such a curve would be

$$Y_n = Y_0 - nD$$

whereby  $Y_n$  is the height of any contraction,  $n$  the number of contractions which have preceded it,  $Y_0$  the height of the first contraction, and  $D$  the constant difference. The fatigability of the muscle is evidently indicated by the constant  $D$ , which is characteristic of each frog, and would have to be determined by experiment.

Ergographic curves are almost never straight lines, hence one constant is in their case insufficient. But we know that fatigue may be caused in several ways, *e.g.* by the using up of the muscle's reserves or by the action of toxic products, and by introducing several constants into the equation we may find it possible to distinguish the portion of the total effect due to each of the several causes.

By far the commonest type of ergogram is that in which the

curve descends rather quickly at first, then continues for some time almost parallel to the axis of the abscissa, to which it finally falls more or less rapidly.

The writer points out that hitherto in ergographic studies the *form* of the curve has been greatly neglected. The difficulty of defining it is indeed serious, so much so that Binet and Vaschide have proposed to substitute for it the height to which the weight has been raised by the middle contraction. This result would serve to show whether the initial force has been well maintained, or has diminished rapidly.

By Kraepelin first, and subsequently by Dr Ioteyko herself, two elements in the curve have been distinguished, viz. the *number* of the contractions and their *height*, and it has been shown that the variations of these two factors are not always parallel to one another.

I. *First Attempts at Mathematical Determination.*—As these early attempts did not lead to results of any permanent value, and as their interest is mainly historical, I shall not attempt to summarise the methods described.

II. *General Equation to the Curve of Fatigue.*—The starting-point of the investigation is the fact established by Mosso, that for the same individual in the same circumstances the ergogram remains the same. The best way to demonstrate this fact is to take two tracings at the same sitting, sufficient time for the muscle to rest being, of course, allowed between the tracings. The second ergogram will be found to be an exact duplicate of the first. This regularity in the curve indicates that some law connects its variables, *i.e.* the time (or abscissa) and the height of contraction (or ordinate). The problem is to find this relation.

Any fatigue tracing is taken, and the curve formed by joining the tops of the ordinates is made regular: for this it will be found that only the slightest possible change is necessary. The normal type of curve has only one turning-point, *i.e.* only one point at which the tangent passes from one side of the curve to the other. The equation to such a curve is of the third degree. The following is the equation resulting from general mathematical considerations:—

$$\eta = H - at^3 + bt^2 - ct$$

$\eta$  being the height of contraction at any given moment,  $H$  the maximum initial effort (in millimetres),  $t$  the time (unit of time = 2 seconds),  $a$ ,  $b$ ,  $c$ , constants or parameters.

The meaning of this mathematical law is that the ergographic curve is at any given moment under the influence of three factors (the constants), one of which,  $b$ , tends to raise the curve in proportion to the square of the time, while the others,  $c$  and  $a$ , tend to lower it in proportion respectively to the time itself and to its cube.

The constants have to be ascertained by direct inspection of the tracing. The operation is obviously an easy one: it involves three measurements of ordinate and abscissa, which give us three equations of the first degree. From these it is a simple matter to determine  $a$ ,  $b$ , and  $c$ . The rest of this section of the paper is devoted to the working out of an illustrative example.

III. *The Place of J. Ioteyko's Quotient of Fatigue.*—The "quotient of fatigue" is the ratio existing between the sum of all the contractions and their number. It is thus the average or mean of the ordinates. The object of the present chapter is to point out that the relation of the quotient of fatigue to the time can easily be obtained from the general equation to the curve.

From the definition of the quotient (Q) and summation of the series the writer obtains the following result:—

$$Q = \frac{1}{t} \int_0^t \eta dt = H - \frac{1}{4} at^3 + \frac{1}{3} bt^2 - \frac{1}{2} ct$$

$\int_0^t \eta dt$  representing the area, that is to say, the sum from 0 to  $t$  of the contractions supposed continuous, multiplied by  $dt$ , an interval of time infinitely small.

The quotient of fatigue is thus a quantity strictly linked to the equation, and as it is a quantity which has a physiological meaning, we may conclude that the equation itself is closely connected with physiological characteristics.

IV. *Mathematical Characteristics of the Curve of Fatigue.*—We know that for every individual there exists a characteristic ergogram, amount of mechanical work, and quotient of fatigue. We are now enabled to express the same thing in mathematical language by saying that for every individual the values of the constants remain the same. The vague graphical notion of the *form of the curve* now becomes definite, seeing that four numbers (viz.  $H$  and the parameters,  $a$ ,  $b$ ,  $c$ , see equation above in II.) are sufficient fully to determine it. Individual variations are expressed by differences in these numbers.

An individual's curve may be affected in various ways, as by the ingestion of alcohol, caffeine, and many other substances. Such alteration means a change in the constants, and it ought to be possible to find out which of the constants are affected by these substances, and in what direction. A comparison of the ergograms of the two sexes, of the right and left hands, etc., may also bring to light determining mathematical characteristics, and "in all these cases the differences will be expressed by numbers, which constitutes the greatest perfection to which a science can attain."

MARGARET DRUMMOND.

**THE STRUCTURE AND FUNCTION OF THE TASTE-BUDS OF**  
(14) **THE LARYNX.** JOHN GORDON WILSON, *Brain*, 1905,  
p. 339.

In the larynx the taste-buds, first described by Verson in 1871, are found on the laryngeal surface of the epiglottis and the medial and lateral aspects of the arytenoids. They are usually met with in groups and never closely packed together as in the tongue. They are only found in the squamous epithelium and do not project beyond the surface, but lie in a very shallow saucer-like depression. Each rests on a broad papilla of dense connective tissue.

Like that of the tongue, the laryngeal taste-bud has two types of cell—the spindle-shaped cell and the supporting cell—and also a flask-shaped cavity in its upper segment.

The spindle-shaped cell extends the whole length of the bud, terminating in a long fine process reaching down to the connective tissue of the papilla. Peripherally it is prolonged into a long, fine, hair-like process projecting into the cavity of the taste-bud. The evidence in favour of a special sense function of this cell is based, firstly, on the fact that it colours readily with neurotropic dyes as opposed to a non-colourability of the supporting cell, and secondly, on its morphological character.

The second type of cell—the supporting cell—differs in no way from the corresponding cells in the taste-buds of the tongue.

The mode of ending of the nerves in the taste-buds of the larynx is very complex. There is a sub-epithelial plexus composed of both medullated and non-medullated nerve fibres. From this plexus go to the taste-buds at least two nerve fibres, some medullated and others losing their sheath at a varying distance between the plexus and the bud. These fibres divide repeatedly at the base of the bud and two systems can be made out—those that break up to form a plexus at the base of the bud with prolongations within the bud, and those which go more or less directly round the bud. The latter may branch in the plexus, but there is no anastomosing of the branches as occurs in the fibres of the plexus.

In this complex arrangement three groups of nerve fibres are distinguished—the subgemmal plexus, the intragemmal nerve fibres, and the perigemmal nerve fibres.

The so-called intragemmal nerve fibres are nerves distributed to the epithelial cells which lie between the buds.

The subgemmal plexus lies in the connective tissue and may begin at some distance beneath the bud. It is formed by an anastomosis of one or more fibres which come from the sub-epithelial plexus.

The intragemmal fibres arise both from the subgemmal plexus and directly from the sub-epithelial plexus. They form a branching and interlacing network around the cells, and knob-like endings most marked at the upper third of the bud lying on the cells.

The perigemmal fibres form a plexus of non-medullated varicose nerve fibres which lie around the bud, forming at the apex a circle round the pore. They arise from medullated nerve fibres coming from the sub-epithelial nerve plexus, and losing their sheath before reaching the base of the bud.

The author has never seen any ganglion cells either in preparations by the Golgi method or by vital injection.

The investigations of the author corroborate the statements of Michelson (91) and Kiesow in demonstrating that taste can be perceived in the upper part of the larynx. Taste perception was found to be rapid and sufficiently accurate to differentiate more or less definitely the four primary tastes, though quantitatively and qualitatively inferior to the tongue.

The following hypotheses have been advanced to explain the presence of taste-buds in the larynx: they are a phylogenetic residue, or else they are organs whose chief function is to strengthen the reflexes which close the laryngeal cavity during the passage of food. The author inclines to the view that the reason for their presence in the larynx must be sought in the intricate mechanism of deglutition. The most suitable end organ in protecting the larynx during the passage of food would be one whose natural stimulus is food.

GOLLA.

## **PATHOLOGY.**

### **A CONTRIBUTION TO THE PATHOGENESIS OF PARALYSES**

- (15) **EXPERIMENTALLY INDUCED BY INFECTION WITH PNEUMOCOCCI.** (Ein Beitrag zur Kenntniss der Pathogenese der durch Infektion mit Pneumokokken experimentell erzeugten Paralyse.) LUIGI PANICHI, *Arch. f. exper. Path. u. Pharmak.*, 1905, Bd. liii., S. 339.

SOME of the rabbits exhibited signs of paralysis a few hours after injection with pneumococci, and soon died. In others the paralysis (usually of one or more limbs) was delayed, not commencing until one or more weeks had elapsed after the last injection. In both the acute and chronic cases some of the nerve cells in the anterior and posterior horns of the cord showed signs of degeneration—absence of Nissl's granules, vacuolation of the protoplasm, excentric nucleus, and shrinking of the cell, the pericellular space which was thus enlarged being filled with small round cells.

In the acute cases, hæmorrhages in the cord were infrequent, whereas in the chronic cases small hæmorrhages in the cord were so frequent as to be almost constant. The hæmorrhages were most abundant in the dorsal region, and were more frequent in the grey than in the white matter, but not more numerous in the anterior than in the posterior horn. Degenerated fibres were observed in the peripheral nerves. The hæmorrhages were confined to the cord, there being none in the lungs, intestines, etc.

In one sheep, motor paralysis with anæsthesia of three limbs commenced five days after injection of the virus. The post-mortem findings were thickening of the vessels of the pia, and congestion and hæmorrhages in the cervical portion of the cord. A transient and lesser degree of paralysis was noted in two horses after they had been repeatedly injected with pneumococci.

The author concludes that the paralyzes were the result of hæmorrhages in the cord, and that the latter were due to morbid changes in the walls of arteries and veins.

W. T. RITCHIE.

**AN EXPERIMENTAL RESEARCH ON THE ANATOMICAL  
(16) LOCALISATION OF THE SYMPTOMS OF DELIRIUM  
WHICH ARE PRODUCED BY THE TOXINES OF  
PELLAGRA.** (*Ricerche sperimentali sulla localizzazione  
anatomica dei sintomi di delirio da tossici pellagrogeni.*)  
CARLO CENI, *Annali di Neurologia*, Fasc. 3, 1905.

THIS is a preliminary note of an investigation on the anatomical localisation of the symptoms of toxic delirium which are produced in dogs by the injection of toxins which are derived from some varieties of *Aspergillus* and *Penicillium*, and which give rise to excitement and convulsions. These toxins are quite distinct from the other group obtained from *Aspergillus* and *Penicillium*, which give rise to depressing and paralytic symptoms.

The symptoms appear about half an hour after the injection of the toxins and last for about two hours.

The delirium is accompanied by motor excitement, which consists of marked tremors all over the body, and of convulsions. The symptoms vary in intensity with the virulence of the toxine employed. In these experiments the author has attempted to localise the seat of disturbance by removing various portions of the cortex of the brain, and he has arrived at the following results:—

1. The toxins in pellagra, which possess an exciting and convulsive power, offer a remarkable elective action on the cortical

centres, while they have no functional action on the centres of the spinal cord, of the bulb, or of the basal ganglia.

2. These toxins act by irritating and exciting all the cortical centres, both psycho-motor and psycho-sensory, in a diffuse manner, without showing any special selectivity for one more than for another.

3. The whole of the cerebral cortex of the dog, but especially that of the occipital lobes, is concerned in the production of this delirium.

4. The motor phenomena would appear to be localised in the motor zone, employing that term in its strictest sense.

R. G. ROWS.

**THE PARATHYROID GLANDS IN GRAVES'S DISEASE.** By  
(17) LAURENCE HUMPHREY, *Lancet*, Nov. 11, 1905, p. 1390.

THE object of the author's inquiry was to ascertain whether there is evidence of any pathological change in the parathyroids in Graves's disease, more particularly in the acutely fatal form of the disease. A comparison of the acute symptoms in this condition, with the results of parathyroidectomy in animals, suggests that some changes might be found.

The parathyroids from four cases of Graves's disease were examined. In two cases the parathyroids were extensively infiltrated with fat, which was intruded between the columns of gland cells, and in part only a few scattered epithelial cells were left between the masses of fat, which largely replaced the gland tissue. In the third case there was a lesser degree of fatty infiltration of the parathyroids, and the gland cells appeared to be degenerating. In the fourth case there was no fatty infiltration; the protoplasm of the cells was diminished, and their nuclei large and crowded together. In none of the cases did the parathyroid glands show any signs of compensatory hypertrophy or any evidence that they were becoming more specialised and forming colloid.

In addition to those four cases of Graves's disease, the parathyroids were examined in eighteen cases of death from various causes. The gland tissue exhibited fatty infiltration in five cases.

It would be premature without further observations to regard the partial disappearance of gland tissue in the parathyroid and its replacement by fat as a pathological feature of Graves's disease, or as associated with the severe symptoms in the fatal form of the disease.

W. T. RITCHIE.

**CLINICAL NEUROLOGY.****THE AFFERENT NERVOUS SYSTEM FROM A NEW ASPECT.**

(18) HENRY HEAD, *Brain*, Part XI., Nov. 1905, p. 99.

**THE CONSEQUENCES OF INJURY TO THE PERIPHERAL**

(19) **NERVES IN MAN.** HENRY HEAD and JAMES SHERREN, *Brain*, Part XI., Nov. 1905, p. 116.

THESE two papers are the first of a series of communications, each of which will deal exhaustively with different aspects of a new hypothesis of sensory innervation. This hypothesis is based upon the careful observation of a large number of cases of nerve injury which are here given with all necessary detail. Many of these cases have been watched for a considerable time, during which the individual phenomena were minutely studied. In addition, and of equal importance, observations of a most minute and detailed character have been made on one of the authors (Head), who had, on April 28, 1903, the radial and left external cutaneous nerves of the left arm cut for the express purpose of observing the changes resulting from section and repair of cutaneous sensory nerves. This operation will become a classic, not only for the splendid observations obtained as a result of it, but also because the operation was voluntarily undertaken in the interest of science, entailing no little risk and very considerable suffering.

When a peripheral nerve, such as the ulnar or median, is cut at the wrist, alterations in the sensibility of the skin supplied by the cut nerve at once follows. The following changes can be made out. In a limited area all sensation in the skin is destroyed. This area of total analgesia is therefore solely dependent on the cut nerve for its supply. Surrounding this area is another with a partial loss of sensibility, and with the character of this impaired sensation a goodly portion of these researches is taken up. This area shows the following characteristics: Stimulation by the prick of a pin is felt more severely than over healthy skin. The pain is of a more widespread character, and the site of stimulation is not always accurately localised. The patient cannot tell the two points of a compass when separated by less than 2 centimetres. The power of recognising differences in temperature is so far interfered with that he cannot recognise cold above 22° C. or heat below 40° C. In considering the peculiarities of this area it is not the fibres of the cut nerve that show this modified sensibility, but the fibres of neighbouring nerves that supply certain forms of sensation to this impaired area. It is proposed to designate this limited and altered sensation by the term *Protopathic sensibility*, while the additional elements that go to make up normal sensation are



designated *Epicritic sensibility*. Epicrotic sensibility presents very well marked and definite qualities, such as greater sensitiveness to touch, as shown by the recognition of gentle stimulation, as by cotton wool, the more precise and definite sense of localisation, the perception of the two points of the compass as distinct when separated by less than 2 centimetres, and the discrimination of fine grades of temperature.

Epicrotic sensibility is not only an addition to the protopathic sensibility, but its presence has a remarkable inhibitory or modifying effect upon the protopathic sensibility, inasmuch as the pain felt by a prick is less severe, and the radiation of the pain and of cold is much less extensive, than when protopathic sensibility is alone present.

There is a third form of sensibility, namely, a *deep sensibility*, which responds to pressure and to movements of joints. It is capable of evoking pain when the pressure is excessive or when a joint is injured. It is best demonstrated in a part where the skin is totally analgesic owing to division of a purely sensory nerve. For the reason that it is thus present when the skin is totally insensitive, it is inferred that the sensory nerves supplying deeper structures, as the muscles, tendons, bones, and joints, reach their destination, not by the purely sensory nerves, but by the nerves supplying the muscles. Support for this inference is found in the facts that Sherrington has found afferent fibres in the nerves supplying the muscles, and that section of motor nerves deprives the muscles and deep structures of all sensation. If an injury severs the ulnar nerve at the elbow before the muscular branches are given off, then deep sensibility of certain of the fingers is destroyed. But if the ulnar is cut at the wrist, then the deep sensibility of the fingers persists. If at the wrist the injury severs the tendons as well as the ulnar nerve, then the deep sensibility of these fingers is lost. Hence it is argued that the fibres supplying the deep sensibility of those fingers reach their destination by running along the tendons.

The sensory mechanism in the peripheral nerves is thus found to consist of three systems: (1) deep sensibility, (2) protopathic sensibility, (3) epicrotic sensibility. A vast amount of information is to be gained by studying the phenomena that accompany the gradual repair of the nerve. There is invariably a very considerable delay after complete division before sensation begins to be restored, and the restoration of sensation is pursued in a very orderly manner. The first sign of recovery is shown by a diminution in the size of the analgesic area due to the gradual spreading over it of protopathic sensibility. This proceeds until the whole analgesic area is covered by protopathic sensibility. The time that elapses from the division of the nerve to the beginning of recovery usually

extends to two or three months, while it may be more than six months before complete restoration of protopathic sensibility takes place. After protopathic sensibility has been completely restored, there is often a pause of two or three months before any sign of improvement takes place in the epicrotic sensibility. When it does begin to recover, the first sign is shown in a blurring of the margin that separates the protopathic sensibility from the normal sensibility of the skin. There is a simultaneous return of all the different forms of sensation by which epicrotic sensibility is recognised—of light touch, of better localisation, of appreciation of finer grades of temperature, and so on. This recovery is very gradual, and in most cases takes more than a year to complete. Any insurance to the healing of the wound delays the return of the higher forms of sensation.

If a nerve has been bruised or incompletely divided, it may fail to conduct impulses, and the resultant loss of sensation may in the first instance resemble that which follows complete division. Recovery, however, in such cases pursues a course different from that which follows complete division of the nerve. Thus at the end of a period which varies with the extent of the injury, the sensibility to prick and to light touch return simultaneously, and as recovery progresses protopathic and epicrotic sensibility return together. By observing the form of recovery, one can tell whether the injury has completely severed the nerve or merely injured it.

A difference in the distribution of protopathic and epicrotic sensibility is observed depending partly on the distance of the cut nerve from the spinal cord. Thus section of a peripheral branch of a nerve near its final distribution presents a different result from section of a nerve near its exit from the spinal cord. If the forearm and hand be divided into a pre-axial (radial) and post-axial (ulnar) half, it is found that the nerves supplying one of these halves overlap only to a slight extent the areas supplied by the nerves of the other half, while the peripheral branches that supply one of these areas overlap among themselves to a very considerable extent. Thus section of the internal cutaneous high in the arm produced an area of total analgesia embracing the greater portion of the ulnar half of the forearm and hand, while at the same time the epicrotic sensation was lost over the remaining portion of the ulnar half of the forearm and hand. This shows that there is very little overlapping of the nerves supplying the radial half of the forearm with the field supplied by the internal cutaneous. Division of one of the two branches of the internal cutaneous presents a very different result—the overlap being so great that little or no analgesia results from section of one branch only.

Injury to the cords of the brachial plexus produces not only

very considerable changes in the sensibility of the parts supplied by the nerves constituting the cord, but they sometimes show a great difference in the relationship of protopathic and epicrotic sensibility compared to what happens in sections of a peripheral nerve. Here the areas of protopathic and epicrotic sensibility are nearly co-extensive.

A further difference in the relationship of these two forms of sensibility is shown when the posterior nerve roots are cut. In two cases division of several posterior nerve roots resulted in the loss of protopathic sensibility over an area greater than that of epicrotic sensibility, that is to say there was an abolition of the sensation to prick over an area larger and more sharply defined than that which became insensitive to light touch. Moreover, this insensibility to prick was accompanied by an inability to appreciate temperature below 15° C. and above 60° C., although 40° C. and 23° C. appeared definitely warm and cool.

In addition to the description of the three forms of sensibility, a number of other points bearing on the distribution of the nerves and on the peculiarities of the various sensations are discussed. The nerve supply of the forearm as shown by changes in sensation due to section of separate nerves is compared with the usual anatomical description. The nerve supply of the leg is studied from the same point of view. The characteristic features of deep sensibility, sensations of heat and cold, the compass test, hair sensibility, hyperalgesia, changes in the skin and nails after injury to nerves, are all minutely detailed.

The papers are illustrated by a large number of excellent drawings that show at a glance the areas affected, and a large number of illustrative cases are given in the text and in the Appendix.

JAMES MACKENZIE.

**A CASE OF NEURITIS, POSSIBLY ATTRIBUTABLE TO  
(20) WORKING WITH ARTIFICIAL MANURES. (Sur un  
Cas de Névrite du peutêtre à l'Usage d'Engrais Artificiels.)  
BABINSKI, *C. R. Soc. de Neur. de Paris*, Jan. 12, 1905.**

A SYMMETRICAL bilateral paralysis of the extensors of the wrist and of the proximal phalanges in an agricultural labourer. When one electrode of a galvanic circuit is placed on the back and the other on the middle of the posterior aspect of the forearm, closure is accompanied by abrupt flexion of the hand and fingers, which is immediately followed by a somewhat slower contraction of the extensors corresponding. The explanation probably is that the former group comes within the field of electrical excitation, and is the first to react, inasmuch as the extensors are deprived of the

stimulating influence of the nerves that supply them, and therefore react more slowly.

It is possible that the presence of arsenic and lead in the impure superphosphates which compose the artificial manure with which the patient has to deal may have been the exciting cause of the neuritis.

S. A. K. WILSON.

#### **MULTIPLE SCLEROSIS IN THE GUISE OF TRANSVERSE**

- (21) **MYELITIS.** (Multiple Sklerose unter dem Bilde der Myelitis transversa.) MORAWITZ, *Münch. med. Wchschr.*, Nov. 7, 1905, p. 2170.

THE writer records two cases of undoubted disseminated sclerosis which for a time during their course showed the symptoms of transverse myelitis. He remarks that, out of the immense variety shown by cases of disseminated sclerosis in their earlier stages, only a limited number have been recorded as belonging to this type, and he gives seven references to other cases.

In one case, that of a farm worker seventeen years old, the patient suddenly developed, at the age of fifteen, after a chill, a weakness and numbness of the right side of the body. This passed away in a fortnight, and left the patient quite healthy till two years afterwards (June 1902), when he sustained a severe fall upon the back of the head, followed a week later by a thorough wetting. Soon afterwards a feeling of weakness and numbness in both legs appeared and passed rapidly into complete spastic paraplegia, the legs being absolutely helpless, the patient unable to pass urine or faeces, a large bed sore forming, and partial anæsthesia being present below the level of the ninth dorsal spine. Nevertheless this condition gradually passed off, and in February 1903 the patient had lost his paralytic condition and presented only somewhat active reflexes. In May 1903 he was able to resume light work in the fields. In May 1904 he complained again of numbness in arms and legs, and on examination again showed spastic paresis of the lower limbs, with diminished sensibility. By October 1904 these symptoms had developed into those of an undoubted multiple sclerosis, including double optic atrophy, scanning speech, intention tremors, and bladder and rectal trouble.

In the other case a man, aged fifty-two, came under observation in May 1905, with the history that since 1884 he had suffered now and then from pains and weakness in the right arm and leg sufficient to confine him to bed for a month at a time. In January 1904 the weakness of the right leg was very marked, and the left leg gradually became affected. The weakness increased till he became unable to move the limbs or voluntarily pass his evacuations. In May 1905 the lower limbs were rigid, the knee- and

ankle-jerks much increased, the Babinski sign present in both feet, but there was no affection of the cranial nerves nor of the fundus oculi. The patient died in August 1905, and the post-mortem examination showed grey sclerotic patches scattered through the cord from the middle of the dorsal region downwards, as well as in the cerebrum, though there were none in the cerebellum, pons, or medulla.

JOHN D. COMRIE.

#### **MINOR AIDS IN THE EXAMINATION OF CASES OF BRAIN**

(22) **DISEASE.** (*Kleine Hilfsmittel bei der Untersuchung von Gehirnkranken.*) H. LIEPMANN, *Deutsche med. Wchnschr.*, Sept. 21, 1905.

LIEPMANN promises a more complete work on the technique of clinical examination in cases of brain disease, but in the meantime suggests various methods which enable one to carry an examination a little further than is frequently done. 1. A patient may react so little to questions and demands that the physician is in doubt as to whether there is not a general dulling of consciousness or marked dementia. If one by means of gesture can bring the patient to imitate movements, it is obvious that the defect is circumscribed and not general. 2. Sometimes it is difficult to determine whether speech is understood by the patient: *e.g.* a motor aphasic cannot give verbal answers, and one has to depend upon his reactions to spoken orders. Here there is a source of misinterpretation, for a patient whose right-sided hemiplegia is due to a cortical focus, especially if aphasic, frequently shows apraxia in the left hand as well. A false reaction to a spoken order may depend, therefore, on the apraxia and not on failure to understand the order. Where apraxia is present, it is not as a rule general; one must seek out a non-apractic muscle group, and by the reactions of this group come to a conclusion about the understanding of spoken orders; the muscles of facial expression often escape the apraxia. Even if the apraxia be general, one can still get the information sought by using the emotional reactions of patient; if one ask patient an insulting question or a grotesque and amusing one, his emotional reaction enables one to determine how far it is understood. 3. Where the patient cannot read, the question arises whether it is due to mind-blindness or to a defect of primary identification or perception. There are various methods of determining the extent of the visual field, the condition of the colour sense, visual acuity. If a patient be asked to pick up peas spread upon his table, and if he fails to pick up those on one side of the visual field, hemianopia is probably present. The introduction of an object of special interest to the patient into his visual

field is also a useful test. Visual acuity can be tested by means of figures, or by asking the patient to pick up small particles of a substance. 4. Perseveration in reactions is frequently misleading: the first reaction of a series is the only one to be used if one be testing a patient with perseveration for mind-blindness. 5. To test stereognosis is not enough: one should use a variety of articles of various texture and surface qualities, and see how far patient can still elaborate the sensation into a complex idea. 6. To test for apraxia, the following series of reactions should be examined: (a) to the order of a simple movement, *e.g.* to spread out fingers; (b) to the order of an expressive movement, *e.g.* to salute; (c) to the order to demonstrate some purposeful movement, *e.g.* how one swims; (d) to the order to show how one uses certain objects, *e.g.* smokes a cigar.

One must include both transitive and intransitive movements, because the "agnostic" who does not recognise objects, *e.g.* a mind-blind patient, can clench his fist correctly when asked, but cannot show how to smoke a cigar.

It is very important to observe the spontaneous behaviour of apractic patients.

C. MACFIE CAMPBELL.

#### LACUNAR AND MYELOPATHIC PARAPLEGIA IN THE OLD.

- (23) (Les Paraplégies d'origine lacunaire et d'origine myélopathique chez les vieillards.) LEJONNE et LHERMITTE, *Arch. Gén. de Méd.*, Nov. 28 and Dec. 5, 1905, pp. 3009 and 3073.

IN the progressive paraplegia that so frequently attacks old people of sixty years and upward, it seems to be possible to distinguish two types, differing alike in their pathological features and their clinical expression. One group connects those forms which are the result of lacunar disintegration in the basal nuclei—a condition fully authenticated by Pierre Marie, Ferrand, and Catola—and which may therefore be described as lacunar paraplegias, and the other includes the myelopathic paraplegias, due to diffuse polyfascicular sclerosis.

The former is commonly accompanied by indications of imbecility and dementia, is of essentially rapid evolution, and is based on definitely marked pyramidal degeneration; this involves the direct and the crossed tracts alike, and is often associated with some sclerosis in the posterior columns. In the latter category the explanation of the symptoms is a diffuse and disseminated sclerosis of the posterior, and more especially of the lateral, regions of the cord, not at all systematised, or obeying any obvious law of distribution. The fact that examination of the cords of old subjects free from all indication of nervous lesions in life often reveals unmistakable arterial disease, is sufficient to illustrate the

discordance between the alterations of the vessels and the extent of the sclerosis, and to warrant the use of the term "polyfascicular sclerosis" in preference to medullary arterio-sclerosis.

As far as the pathogeny of the two conditions is concerned, it is unwise to speak dogmatically. There seems to be little doubt, however, that the systematised pyramidal degeneration of lacunar paraplegia is consecutive to a process of subacute vaginitis around the striate arteries of the basal ganglia, but in the myelopathic type there does not appear to be any relation between irregular patches of diffuse sclerosis and perivascular disintegration. The thickening of the adventitia sometimes met with in the blood-vessels round the patches is rather the result than the cause of the sclerosis.

S. A. K. WILSON.

#### **LATENT FORMS OF AFFECTIONS OF THE PYRAMIDAL**

- (24) **SYSTEM.** (*Formes Latentes des Affections du Système Pyramidal.*) BABINSKI, *C. R. Soc. de Neur. de Paris*, Jan. 12, 1905.

THE presence of a single or double extensor response may be the sole persistent indication of a preceding affection of the pyramidal tracts, and it may occur in the complete absence of any clinical evidence pointing to the existence of organic disease. The "fan sign" may be taken to signify that there is some disorder of the pyramidal system; it is more common in infantile than in adult hemiplegia, more usual in hemiparesis than in hemiplegia, and more frequently met with in spinal than in cerebral paralysis.

S. A. K. WILSON.

#### **"BLUE DISEASE"—RETINAL CYANOSIS—HEMIPLEGIA FOLLOWING**

- (25) **LOWING ON WHOOPING-COUGH.** (*Maladie bleue—Cyanose de Rétines — Hémiplegie consécutive à une Coqueluche.*) BABINSKI and TOUFESCO, *C. R. Soc. de Neur. de Paris*, Nov. 3, 1904.

A SHORT report of a ten-year-old boy, who at the age of three suffered from a severe attack of whooping-cough of five months' duration, in the course of which he had an ictus, remaining unconscious for a quarter of an hour. A left hemiplegia followed. From that time it was noticed that even when at rest he showed a bluish coloration of his skin and mucous membranes, a condition which any exertion markedly accentuated. Examination of the fundus revealed deep cyanosis of the retina and underlying structures, with great tortuosity of the veins, which were of a blue-violet colour.

S. A. K. WILSON.

**ATROPHY OF THE CELLS OF PURKINJE.** (*Atrophie lamellaire*  
(26) *des cellules de Purkinje.*) ANDRÉ THOMAS, *Rev. Neurol.*,  
Sept. 1905, p. 917.

THE author describes the case of a woman, æt. 54, who had previously suffered from erysipelas, typhoid, and syphilis. Her feet had been deformed as long as she could remember [talipes equino varus], greater on the right side than on the left. For four years before her death she had developed signs pointing to an affection of the cerebellum. On examination she had slight nystagmus on extreme lateral deviation of the eyes. The upper extremities were normal. The lower extremities were deformed as above mentioned, and the muscles were hypotonic, though not wasted. There was marked ataxia of the legs and a tremor simulating that of disseminated sclerosis. She walked on a wide base with marked oscillations of the trunk and a cerebellar gait. Rombergism was present. The knee-jerks were increased, especially the right, the ankle-jerks were diminished, and the plantar reflexes were extensor in type. There was no diadococinesia. The post-mortem examination revealed no gross lesions. On microscopic examination the following changes were discovered:—

1. *In the cerebellum (stained with Picrocarmine).*—In certain lamellæ, Purkinje's cells had completely disappeared, while in the immediate neighbourhood they remained normal.

Where the Purkinje's cells had disappeared their place was taken by a thick matting of neuroglia fibrils, which in some places invaded the molecular and granular layers. In other places there were a number of neuroglial nuclei which formed a distinct layer between the molecular and the granular layers.

In the molecular layer, numerous amyloid bodies were found, and the cells in the granular layer were diminished in number and stained unequally.

The vessels, the meninges, and the central white substance were healthy.

2. *In the spinal cord.*—In the sacro-lumbar region the anterior horns were diminished in size, especially on the right, with diminution of the number of the cells and proliferation of the neuroglia. In the same region there were small foci in which neuroglial fibrils and amyloid bodies were very numerous, and in which there were no cells.

The author points out that such changes as were observed in the cerebellum are by no means rare, and he found them in a case of tabes and of disseminated sclerosis which he examined subsequently. The changes in the spinal cord resembled in many features the changes in the cerebellum, and he thinks that they



were probably of the same nature; the spinal changes occurring in infancy and the cerebellar during the last four years of the patient's life.

T. GRAINGER STEWART.

**PERIPHERAL POST-PARALYTIC HEMISPASM.** (*Hémispasme*  
(27) *facial périphérique post-paralytique.*) CRUCHET, *Revue Neurologique*, Oct. 1905, p. 985.

AFTER reminding the reader of the differences between a tic and a facial spasm, the writer classifies cases of peripheral facial spasm into three groups. Firstly, there is primitive facial spasm, which seems to occur in place of facial palsy and under the same conditions, but is not followed by paralysis; secondly, there is pre-paralytic facial spasm, which precedes an attack of facial palsy; and lastly, there is the post-paralytic spasm, the commonest variety. Of this latter, he describes a case in detail.

The patient, a girl of 11 years, had an attack of left-sided facial palsy four years before. This passed off in three months. About a year later, a tonic spasm was observed to be present in the orbicularis oculi, narrowing the palpebral fissure on that side. This gradually increased in intensity. After two years, clonic muscular twitchings were observed on the left side of the face, beginning at the left angle of the mouth, later affecting also the eye and the chin, and causing a dimple on the left side of the chin. In addition, emotional movements produced a tonic over-action of the muscles on that side.

Cruchet considers his case an example of spasm preceding contracture. The notes of the case, however, would tend to show that the two conditions developed simultaneously.

PURVES STEWART.

**SPASM OF THE RIGHT TRAPEZIUS AND FACIAL TIC.**  
(28) (*Spasme du Trapèze Droit et Tic de la Face.*) BABINSKI, *C. R. Soc. de Neur. de Paris*, July 6, 1905.

IN opposition to the views of Pitres and Cruchet, who would limit the application of tic to abrupt muscular twitches of brief duration, Babinski holds that the contraction need be neither brief nor abrupt, thereby adopting the theory of Brissaud and Meige. The case reported presented the interesting combination of trapezius spasm—for some of the movements were such as could not have been imitated by an effort of the will, and they continued during sleep—with a true grimacing facial tic, which occurred chiefly, though not exclusively, in the exercise of such functions as speaking or swallowing.

S. A. K. WILSON.

**PERIPHERAL FACIAL HEMISPASM.** (*Hémispasme facial périphérique.*) J. BABINSKI, *Nouv. Icon. de la Salpêtrière*, juillet-août 1905, p. 419.

THE patient here described suffered from a bulbar lesion, apparently bilateral, though more marked on the left side. His chief symptoms were vertigo, latero-pulsion to the left, a bilateral extensor plantar response, right-sided combined flexion of leg and pelvis, paresis of the right vocal cord, left-sided hemiatrophy of the tongue, and left-sided hemiparesis of the face. This latter phenomenon was succeeded by a left-sided facial hemispasm.

This hemispasm consisted of a series of brisk twitches, short and in rapid succession, finally culminating in a tonic spasm lasting several seconds, similar to what might be produced by a series of faradic shocks, gradually increasing in frequency. These attacks were uncontrollable by the patient, and were more frequent when he was fatigued. The individual spasms began as fibrillary contractions, limited to a few muscular fibres at first, and producing deformities which could not be imitated voluntarily on the healthy side, *e.g.* puckering of the chin, deviation of the tip of the nose. Moreover, contradictory facial muscles were often thrown into contraction simultaneously, *e.g.* the platysma and the levator labii superioris, the orbicularis oculi and the frontalis. This characteristic distinguishes facial hemispasm from facial tic, the latter being psychic in origin and capable of voluntary imitation. Hemispasm can persist during sleep, whereas a tic ceases then.

Babinski compares the facial spasm of *tic douloureux* with that of facial hemispasm of motor origin, the former being due to irritation of the afferent limb of the reflex arc, the latter to irritation of the efferent nerve.

PURVES STEWART.

**SYNERGIC PARADOXICAL CONTRACTIONS FOLLOWING PERIPHERAL FACIAL PALSY.** (*Contractions "synergiques paradoxales" observées à la suite de la paralysie faciale périphérique.*) H. LAMY, *Nouv. Icon. de la Salpêtrière*, juillet-août 1905, p. 424.

UNDER the above title the writer describes the well-known clinical phenomenon that, in many cases of imperfectly recovered facial palsy, the patient, when he succeeds in innervating the paresed side of the face, has imperfect control over the muscles and throws into contraction others that he had not intended.

Lamy's patient (who also showed the familiar involuntary fibrillary twitchings of incompletely recovered facial palsy) was a

man of over 60 years of age who had suffered since childhood from facial palsy of the right side. The muscles most deficient in voluntary contraction were the *frontalis*, *zygomatici*, and *levatoris labii superioris*. Yet when he closed the eyes, or still more, when he closed the eye of the paralysed side alone, all these muscles above-mentioned were thrown into strong contraction. He therefore terms the phenomenon "paradoxical synergy," inasmuch as the contraction of these muscles is illogical and absurd, for the *frontalis* is normally an opponent of the *orbicularis oculi*.

Lamy recalls a similar involuntary movement of the shoulder which often accompanies facial movement in cases of facio-accessory anastomosis. And he would explain the "paradoxical synergy" on the same lines, as being due to innervation of the *frontalis* and *zygomatici* by an abnormal part of the facial nerve, viz. its *orbicularis* branch.

PURVES STEWART.

**BLOOD - PRESSURE AND NEURASTHENIA.** (*Blutdruck und*  
(31) *Neurasthenie.*) S. FEDERN, *Wiener med. Wchnschr.*, Nov. 4, 1905, p. 2157.

IN this paper the writer replies to certain critics who have thrown doubts upon his theory that the symptoms of neurasthenia are attributable to a constantly heightened blood-pressure. The work to which the writer chiefly animadverts is contained in papers by Haskovec (*Wien. med. Wchnschr.*, Nos. 11-17, for 1905). The heightened blood-pressure which the writer states that he finds in neurasthenic persons is referred by him in general to peripheral irritation of the splanchnic nerves brought about by a condition of intestinal atony, though he allows that it may be caused by disturbances of other organs. He adduces no new facts in support of his theories.

JOHN D. COMRIE.

**HYSTERICAL TORTICOLLIS.** (*Torticollis hystericus.*) KOLLARITS,  
(32) *Deut. Ztschr. f. Nervenheilk.*, H. 5-6, Nov. 1905, p. 413.

DETAILS of three cases of spasmodic torticollis are given, and references to three others previously described. In all six a neuropathic heredity was evident, although it is stated that Jendrassik, who also reported on the second series, found hysterical stigmata in one only. The direct cause of the torticollis was in (1) tremor of the head; (2) paræsthesia in neck; (3) an insignificant blow which directed attention to the neck. In none of the six was the spasm confined to the region of the *accessorius*; other muscles were always involved, giving rise to

the characteristic co-ordinated movements or spasmodic attitudes. All had discovered some mode of preventing the spasm, which they exercised without any expenditure of strength and which could only have had an auto-suggestive influence. Good results, though not perfect "cures," were obtained in three cases by hypnotism and exercises; one was improved by suggestive methods; in the other two there was no improvement. The author concludes that every spasmodic torticollis is a mental one, and as such, a symptom of hysteria, although possibly a mono-symptomatic hysteria. The treatment can only be by suggestive methods.

Some good photographic illustrations of the condition are given.

J. H. HARVEY PIRIE.

**A FURTHER CONTRIBUTION TO THE PATHOGENESIS OF  
(33) EXOPHTHALMIC GOITRE.** ALFRED GORDON, *New York  
Med. Journ.*, 1905, Nov. 4, p. 955.

THE author describes a case which he regards as a valuable contribution to the subject of the nervous origin of exophthalmic goitre, and especially to its probable origin in the medulla.

A middle-aged woman suddenly noticed that she could not raise her eyes in the normal manner; on the following day there was inability to turn the left eye externally. A few days later prominence of both eyeballs gradually ensued. A week later the neck became enlarged anteriorly, and at the same time cardiac palpitation and tremor of the hands made their appearance. On examination there was found exophthalmos, von Gräfe's sign, goitre, tachycardia, and tremor, with paralysis of the third, fourth, and sixth cranial nerves, unequally distributed on the two sides.

W. T. RITCHIE.

**SOME INVESTIGATIONS ON THE NERVOUS MANIFESTATIONS  
(34) OF ACUTE RHEUMATISM.** F. J. POYNTON and ALEXANDER  
PAINE, *Lancet*, Dec. 16, 1905, p. 1760.

IN this paper, which was read before the Neurological Society of the United Kingdom, the authors refer in the first place to their well-known views as to the etiology of acute rheumatism. They firmly believe that rheumatic fever is a definite disease, and, further, that the diplococcus rheumaticus which they have described is a cause of the disease. The authors are of opinion that they have satisfied Koch's postulates; on the other hand they "have never maintained that this diplococcus was specific except in so far that it is, in our opinion, *the only bacterial cause of a specific disease.*"

It remains for those who oppose the view that rheumatic fever is the result of an infection, to demonstrate some infective cause and to explain the coincidence that a bacterium which has been found in the arthritis, endocarditis, pericarditis, subcutaneous nodules, pleurisy, pneumonia, peritonitis, and nephritis of rheumatic fever, is able to produce similar lesions in animals. The diplococcus is found with remarkable constancy in suitably chosen cases. It is very difficult to discover if looked for in unlikely places, such as the blood and arthritic exudations. The nature of the organism is a question of secondary importance as compared with the question as to whether it is the cause of acute rheumatism. A specific test has not yet been found for the organism, but this, the authors maintain, in no way alters their conclusions above mentioned. The peculiarities of the diplococcus are then described.

In the second part of the paper the authors summarise their reasons for believing that rheumatic chorea is a local infection of the nervous system, and that most of its symptoms are the result of a slight meningo-encephalitis and possibly meningo-myelitis.

1. They have isolated and cultivated the diplococcus from the cerebro-spinal fluid in four cases of fatal rheumatism, in three of which there was chorea at the time of death.

2. They have produced twitching movements, arthritis, endocarditis, and pericarditis by intravenous injections of the diplococcus into rabbits.

3. They have demonstrated the presence of diplococcus three times in the cerebral pia mater and once in the brain from cases of chorea.

4. Also in the brain and pia mater of the rabbit that had shown twitching movements.

The third section of the paper is devoted to Rheumatic Meningitis, to which it is to be noted they attach a mark of interrogation. The case was that of a boy, aged 13, admitted to hospital with what appeared to be an ordinary attack of rheumatic fever. Under treatment all pain and swelling had disappeared within four days. For three weeks uninterrupted convalescence continued. Then he complained of headache, he was sick, and his temperature rose to  $102.4^{\circ}$ . Later he became drowsy and collapsed, cyanosed and pulseless. On the third day after the temperature had risen he became unconscious, with fixed dilated pupils and general muscular rigidity, alternating with flaccidity, and died the same evening. The most probable diagnosis appeared to be cerebral rheumatism.

At the necropsy made fifteen hours after death, early endocarditis of the mitral valve with a slight left-sided pleuro-pericarditis was observed. In no part of the body could any focus of suppura-

tion be seen. A meningitis almost entirely basic in distribution was found. A good deal of turbid fluid was present, with flakes of exudation. There was also a general spinal meningitis. Minute diplococci were found in pus from the cerebro-spinal fluid. Cultures from the cerebro-spinal fluid in bouillon, milk, and glycerine agar contained a pure growth of the diplococcus. Two rabbits which were inoculated died in twenty-four hours. The diplococcus was isolated from the blood, and an intravenous injection into a rabbit was followed by an arthritis. The diplococcus was recovered from the damaged joints. The authors believe that this organism was the diplococcus rheumaticus, notwithstanding that they observed capsulation in those obtained from the rabbits' blood, a feature which they admit has hitherto been regarded as a distinctive feature of the pneumococcus.

Rheumatic meningitis the authors would place midway between the slight local rheumatic lesions, which they believe exist in chorea, and acute fatal rheumatic hyperpyrexia, which they regard as an acute rheumatic toxæmia.

EDWIN BRAMWELL.

**A FURTHER CASE OF HEMICRANIOSIS.** (*Sur un Nouveau Cas (35) d'Hemicraniose.*) PARHON and NADJEDE, *Rev. Neur.*, Nov. 15, 1905, p. 1017.

Two years ago, Brissaud and Lereboullet described a curious condition of cranial hemihypertrophy, with fronto-parietal and infra-orbital hyperostosis. In one of their cases the autopsy revealed the existence, apart from the bony changes already mentioned, of numerous tumours on the under surface of the dura mater, presenting the histological characters of angiolithic sarcoma. The condition appeared to be the converse of the facial hemiatrophy of Romberg, the trouble being limited to the distribution of the fifth cranial nerve, especially its ophthalmic branch, but on the other hand, it did not correspond to what has been described as facial hemihypertrophy. Some months later, Parhon and Goldstein published a case in which an exostosis about the size of a nut in the right anterior parietal region was found post-mortem to be associated with a similar growth on the under surface of the skull, and this in its turn exactly fitted into a depression in another tumour, of the dimensions of a mandarin orange, which was growing from the dura and pressing into the greater part of the motor area and frontal lobe. The latter was an angiolithic sarcoma. Is this an instance of one tumour serving as a *point de départ* for another tumour of a different nature?

The authors report an additional case, the interest of which is

unfortunately neutralised by the paucity of the clinical information and the absence of all reference to the relation between the clinical and the pathological data. At the section on an old woman with left hemiplegia, a bony excrescence was found of the size of a nut, springing from the middle and posterior part of the right frontal bone, and dimpling neatly into a second tumour of the size of an orange, which grew from the dura and was burrowing into the right prefrontal lobe. This latter neoplasm was an angiolithic sarcoma. The authors are content to record the facts.

S. A. K. WILSON.

**ON THE QUESTION OF INTENTION TREMOR IN CHILDREN.**

(36) (*Zur Kasuistik des Intentionstremors bei Kindern.*) URBACH,  
*Deut. med. Wchnschr.*, Oct. 19, 1905.

THE symptoms of four children showing marked tremor upon purposive movements are recorded. The first two cases are those of a brother and sister, aged seven and five years respectively. Both showed tremors markedly in the hands and feet when an effort was made, but neither presented any other symptoms except cramps in the hands and feet, of which the girl complained. On careful examination her urine was found to contain a discoverable though small quantity of lead. These two cases of tremor the writer attributes to lead poisoning, though on careful examination of the children's surroundings at home he was quite unable to discover the source of the lead. The tremors in both children disappeared in the course of a year. Of other two similar cases that he records the writer attributes one to probable lead poisoning, though no lead was discoverable in the urine; while the other he regards as an example of hereditary tremor.

A large portion of the paper deals with the diagnosis of tremors generally in children, the conditions discussed as likely to cause tremor being multiple sclerosis, pseudo-sclerosis, cerebellar ataxia, tumour of the cerebellum or of the optic thalamus, exophthalmic goitre, toxic conditions, hereditary tremor, and hysteria.

JOHN D. COMBIE.

**THERMO-ASYMMETRY OF BULBAR ORIGIN. (*Thermo-asymetrie***

**(37) *d'origine bulbaire.*)** BABINSKI, *C. R. Soc. de Neur. de Paris*,  
May 11, 1905.

A HEALTHY man, thirty-five years of age, was suddenly seized with vertigo and staggering of some fifteen minutes' duration. The next day he noticed that the whole of the right half of his body was colder to the touch than the left, and felt colder. On the next

day a friend remarked that his left eye was smaller than the right, and the conjunctiva was injected. When examined at the hospital twenty-four hours later, he was found to present a left palpebral aperture narrower than its fellow, with enophthalmos and myosis on the same side: the temperature of the left half of the body was more than a degree above that of the right, which showed a marked extent of syringomyelic dissociation of sensation, the face alone not participating. At the end of three weeks the symptoms had practically disappeared.

Evidently, then—for the lesion must have been situated in the left half of the medulla or ponto-medullary region—a bulbar lesion may give rise to vaso-motor and thermic disturbances of hemiplegic distribution, without causing an ordinary motor hemiplegia. Such a disorder may be named thermo-asymmetry. A convenient test for its demonstration is to immerse the hands in cold water for a few minutes, and compare the after-effects.

S. A. K. WILSON.

## TREATMENT.

### OUTLINES OF THE TREATMENT OF MENTAL DISEASES.

(38) (*Grundzüge zur Behandlung der Geisteskrankheiten.*) S. BINSWANGER (of Jena), *Deutsche med. Wchschr.*, March 9, 1905.

IN the treatment of mental diseases the physician must keep in mind the individual reaction: this is especially true in the prophylactic measures to be taken with regard to children with a psychopathic disposition. Binswanger accentuates the necessity of a well-regulated life for these children, and calls attention to the main points which should guide the physician.

He discusses the treatment of the initial stages of mental disease, and calls attention to two main groups of phenomena—the anomalies of mood and the signs of mental fatigue. Where the patient is boisterous and excited, hospital treatment is imperative; where he is quiet and depressed, he may be treated at home, but only if in good circumstances and on condition that there shall be sufficient skilled nursing and ample accommodation. In depressed conditions, with much anxiety, Binswanger recommends a systematic treatment with opium.

Where patients present a primary blocking of thought with a want of emotional reactions, one must distinguish between the beginning of a stupor and the first stage of a severe depression. In the former, treatment should be stimulating; in the latter, opium and sedatives are indicated. One must be careful not to



overdo treatment in the stuporous cases—massage, gymnastics, electricity, hydrotherapy; for in exhaustion and toxic cases, stupor may pass into excitement, and stimulating treatment must cease as soon as irritative symptoms appear. In conclusion, Binswanger makes a few remarks on the general treatment adopted in modern psychiatric hospitals.

C. MACFIE CAMPBELL.

**BALNÉATION ET HYDROTHERAPIE DANS LE TRAITEMENT**

(39) **DES MALADIES MENTALES.** B. PAILHAS. Rennes: F. Simon, 1905, pp. 146.

THIS monograph is an example of a very useful practice which prevails in connection with the French Congresses of Alienists and Neurologists. A specific subject is selected and a certain individual is requested to study it fully and present a report on it to the succeeding congress. In this way much valuable information on current subjects of interest is obtained, and on subsequent publication in book form this information is made available to a much wider circle. The book under notice was presented at the 15th Congress held at Rennes in August last.

From the nature of the subject, not much that is new can be expected in Dr Pailhas' treatise. It is, however, a very useful compendium of the experience of a large number of physicians who have used hydropathy in the treatment of mental disease. It appears to be much more extensively used and trusted as a remedial agent on the Continent than in this country. The reason for this is not by any means clear.

The first chapter contains a short historical review of the subject. The second gives an exposition of the principles on which the treatment is based. They are of various orders, physical, dynamic, chemical, and even biological, but in the main the effect is due to the temperature of the water employed. In a lesser degree the mechanical effect of percussion comes into play in some of the applications. By directly influencing the vascular and nervous mechanisms in the skin a secondary effect is produced in the cerebro-spinal and sympathetic centres. On the whole the effects produced are due to this reaction. The reaction is greater the more the temperature of the water differs from that of the human body, and naturally cold water is the principal agent in obtaining the hydrotherapeutic result. Another general principle enunciated is that the greater the difference of temperature from the normal the shorter should be the period during which it is applied. When the temperature approaches the normal the effect is less and less a reaction, and becomes more and more a sedative one. It is thus possible to choose the hydropathic means best

suited to obtain a therapeutic result. In this connection it would have been desirable to have stated the results of scientific experiments on the effects of various baths on healthy individuals, as has been done with many drugs.

The third chapter contains an account of the various procedures and modes of application of hydrotherapy in the treatment of mental disease. In this, most space is devoted to the prolonged luke-warm bath, which is so strongly recommended by many physicians. It is stated to be easy of application, free from undue risk, and of great value in calming excitement of all kinds. Its effect must, however, be carefully watched in each case. Very hot baths, cold baths, sitz baths, douches, sprays of high, low, and alternating temperature, foot baths, hot and cold packs, Russian, Turkish, and medicated baths are all fully described and the indications for their application given. This information is of such a nature, however, as to be incapable of condensation, and to obtain it reference must be made to the book itself.

The fourth is a most useful chapter, as it gives a general review of the indications for the use of these various remedies in mental cases of all kinds. At the end a few pages are devoted to the author's conclusions. If these are correct, alienists in this country assuredly do not make sufficient use of hydrotherapy as a means of treatment. The reading of this short work might do something to lessen such neglect. The author may be congratulated on having produced a most readable, interesting, and useful book.

JAS. MIDDLEMASS.

**THE ANTITHYREOIDIN TREATMENT OF EXOPHTHALMIC**  
(40) **GOITRE.** (Zur Antithyreoidin-Behandlung der Basedow'schen Krankheit.) A. EULENBURG, *Berl. klin. Wchnschr., Fest-Nummer Carl Anton Ewald*, Oct. 30, 1905.

THE author reviews the literature of the antithyreoidin treatment of exophthalmic goitre, and records seven cases he has thus treated. The initial dose is 10 drops thrice daily. This is increased to 15 drops on the third day, to 20 on the fifth, and to 30 on the ninth day. From the eleventh to the eighteenth day the dose is gradually reduced, then the drug is discontinued for a week, and thereafter it is again given in increasing doses as before.

The conclusion is arrived at that antithyreoidin is of some value in the treatment of exophthalmic goitre, being mainly useful in the treatment of symptoms. Some cases are distinctly benefited, both objectively and subjectively, whereas in other cases the improvement is either slight and transient or there is no benefit obtained.

W. T. RITCHIE.

**CONCERNING SURGICAL INTERVENTION FOR THE INTRA-****(41) CRANIAL HÆMORRHAGES OF THE NEW-BORN. HARVEY**

CUSHING, *Am. Journal of Med. Sci.*, Oct. 1905.

THE author draws attention to the importance of realising the pathology of the so-called "birth palsies" or paralyses of cerebral origin in new-born children. A large number are due to cortical injury during birth, and as most of the cases are not immediately fatal, they are serious only from the distressing late consequences of the injury. The most common cause is a cerebral vascular lesion, generally a hæmorrhage, which is of venous origin, and results from the rupture of those venous radicles of the cerebral cortex which are most weakly supported. It may be due to traumatism at birth or occur as result of severe strain, as by partial asphyxiation from severe whooping-cough or convulsions.

Cushing examined a large number of new-born infants post-mortem, and found that a considerable percentage had died of intracranial hæmorrhage.

The vessels most commonly ruptured are those ascending over the cortex and opening into the superior longitudinal sinus from the mid-cerebral region, where they have no support on leaving the sulci and fissures to enter the sinus. The cranial moulding during parturition or when forceps are applied lays considerable strain on these vessels, and may rupture them where they enter the sinus.

The primary effusion and thickest part of the clot found in these cases is generally median, and consequently implicates primarily and most seriously the centres for the lower extremities.

The effusion is generally limited to one side of the falx, but may be bilateral as in cases of diplegia.

An early diagnosis is important, and in all cases of doubt lumbar puncture should be performed, where a history of severe labour or post-partum asphyxia is given. The fontanelle is often bulging, and may be so light as to show no pulsation. Convulsions appear a few days after birth and are often unilateral in character.

Some children become epileptics often showing a Jacksonian type. Attention is drawn to the importance of recognising these early hæmorrhages, and immediate surgical interference is strongly advised, so as to prevent them reaching the late consequences of the hæmorrhage, so hopeless to treat medically or surgically. He records four cases treated by turning down a parietal flap and exposing the motor areas. The appearances seen were a tense dura mater, often of a plum colour, which on incision revealed a large amount of blood-clot. This was washed out, and the wound closed without drainage.

New-born children stand the operation well. Many of the cases formerly supposed to be infective in origin are probably due to hæmorrhage.

E. SCOTT CARMICHAEL.

# Bibliography

## ANATOMY

- ROTH. The Relation between the Occurrence of White Rami Fibres and the Spinal Accessory Nerve. *Journ. Comp. Neurol. and Psychol.*, Nov. 1905, p. 482.
- FUSARI. Contributo delle terminazioni nervose nei muscoli striati di "Ammonoetes branchialis." *Arch. per le Sc. med.*, No. 5, 1905.
- ARCHAMBAULT. Le faisceau longitudinal inférieur et le faisceau optique central. *Rev. Neurol.*, nov. 30, 1905, p. 1053.
- JOHN TURNER. A Note concerning Mesoglia Cells. *Rev. Neurol. and Psychiat.*, Dec. 1905, p. 773.
- VILLIGER. Gehirn und Rückenmark. Leitfaden für das Studium der Morphologie und des Faserverlaufs. Engelmann, Leipzig, 1905, M. 9.
- PÉREZ. Oreille et Encéphale. Etude d'Anatomie Chirurgicale. Maloine, Paris, 1905, 65 fr.
- BROCK. Untersuchungen über die Entwicklung der Neurofibrillen des Schweinefötus. *Monatsschr. f. Psychiat. u. Neurol.*, Nov. 1905, p. 467.
- WARREN. Development of the Paraphysis and the Pineal region in *Necturus maculatus*. *Am. Journ. of Anat.*, Vol. v., p. 1.

## PHYSIOLOGY

- JOHN B. WATSON. The Effect of bearing of Young upon the Body-Weight and the Weight of the Central Nervous System of the Female White Rat. *Journ. Comp. Neurol. and Psychol.*, Nov. 1905, p. 514.
- JENNINGS. Papers on Reactions to Electricity in Unicellular Organisms. *Journ. Comp. Neurol. and Psychol.*, Nov. 1905, p. 528.
- LAPICQUE. Recherches sur l'excitabilité électrique de différents muscles de vertébrés et d'invertébrés (suite et fin). *Ann. d'Electrobiol. et de Radiol.*, No. 5, 1905, p. 577.
- CLUZET. Loi d'excitation des nerfs par décharges de condensateur (suite et fin). *Ann. d'Electrobiol. et de Radiol.*, No. 5, 1905, p. 595.
- IOTEYKO. Les lois de l'ergographie, étude physiologique et mathématique (suite et fin). *Ann. d'Electrobiol. et de Radiol.*, No. 5, 1905, p. 666.
- COGGI. Sullo sviluppo del sistema nervoso periferico dei Vertebrati e su di una nuova classificazione dei principali organi di senso. *Monitore zool. italiano.*, No. 10, 1905.
- HUBERT RICHARDSON. The Thyroid and Parathyroid Glands. Blakiston, Son & Co., Philadelphia, 1905.
- WILKS. Ambidexterity. Sydney Mayle, London, 1905, 3d.
- HERING. Grundzüge der Lehre vom Lichtsinn. Engelmann, Leipzig, 1905 M. 2.
- MAURER. Untersuchungen zur vergleichenden Muskellehre der Wirbeltiere. Fischer, Jena, 1905, M. 20.
- RAMSTRÖM. Untersuchungen und Studien über die Innervation des Peritoneum der vorderen Bauchwand. *Beitrag zur Anat. u. Entwickl.*, H. 89, p. 349.

## PSYCHOLOGY

- GIMBAL. Les incendiaires. *Ann. Medico-Psychol.*, No. 3, 1905, p. 353.
- LENER. La delinquenza e la pazzia in rapporto alla universalità del fenomeno economico. *Il Manicomio*, Anno xxi., No. 2, 1905, p. 156.
- AMENT. Ein Fall von Überlegung beim Hund. *Arch. f. d. gesamte Psychol.*, B. 6, H. 3, p. 249.

- SEGAL. Die bewusste Selbsttäuschung als Kern des ästhetischen Genießens. *Arch. f. d. gesamte Psychol.*, B. 6, H. 3, p. 254.
- DÜRR. Zur Frage der Wertbestimmung. *Arch. f. d. gesamte Psychol.*, B. 6, H. 3, p. 271.
- BOTTL. Ein Beitrag zur Kenntnis der variablen geometrisch-optischen Streckentäuschungen. *Arch. f. d. gesamte Psychol.*, B. 6, H. 3, p. 306.
- STÖRRING. Experimentelle Beiträge zur Lehre vom Gefühl. *Arch. f. d. gesamte Psychol.*, B. 6, H. 3, p. 316.
- KIESOW. Über einige geometrisch-optische Täuschungen und über sogenannte "frei steigende" Vorstellungen und plötzlich auftretende Änderungen des Gemutszustandes. *Arch. f. d. gesamte Psychol.*, B. 6, H. 3, pp. 289, 357.
- CLIFTON TAYLOR. Über das Verstehen von Worten und Sätzen. *Zeitschr. f. Psychol. u. Physiol. der Sinnesorg.*, Bd. 40, H. 4, p. 225.
- NEISSER. Individualität und Psychose. Hirschwald, Berlin, 1906, M.—60.
- BRAUN. Die religiöse Wahnbildung. Mohr, Tübingen, 1906, M. 1.
- ALT. Über Melodientaubheit und musikalisches Falschhören. Deuticke, Wien, 1906, M. 2.
- KERN. Das Wesen des menschlichen Seelen u. Geisteslebens. Hirschwald, Berlin, 1905, M. 2.40.
- MEDEIROS e ALBUQUERQUE. Que é uma emoção? *Arch. Brasil. de Psychiat. e Neurol.*, Anno i., N. 3-4, 1905, p. 233.
- ROSSI. Dell' attenzione collettiva e sociale. *Il Manicomio*, Anno xxi., No. 2, 1905, p. 248.
- ANGIOLELLA. Sulla genesi biologica del delitto. *Il Manicomio*, Anno xxi., No. 2, 1905, p. 219.
- STIER. Fahnenflucht und unerlaubte Entfernung. Eine psychologische, psychiatrische und militärrechtliche Studie. Marhold, Halle, 1905, M. 3.
- The Feeble-minded Criminal. Leading Article, *Lancet*, Nov. 25, 1905, p. 1556.
- RAYMOND PEARL. Some Results of a Study of Variation and Correlation in Brain Weight. *Journ. Comp. Neurol. and Psychol.*, Nov. 1905, p. 467.
- Brain Weights and Intelligence. Leading Article, *Brit. Med. Journ.*, Nov. 25, 1905, p. 1415.
- JOHN E. ROUSE. Respiration and Emotion in Pigeons. *Journ. Comp. Neurol. and Psychol.*, Nov. 1905, p. 494.

### PATHOLOGY

- ALQUIER. Deux cas d'Hétérotopie du Cervelet dans le canal rachidien. (Soc. de Neurol.) *Rev. Neurol.*, Nov. 30, 1905, p. 1117.
- KATTWINKEL. Sklerose épendymaire en plaques ombiliquées. *Rev. Neurol.*, nov. 30, 1905, p. 1066.
- GUIZZETTI. Pseudo-corporcoli cromatici del cilindrase? *Riv. di Patolog. nerv. e ment.*, Vol. x., f. 10, 1905, p. 473.
- GARBINI. La struttura e la funzione della ipofisi in alcune forme gravi, congenite ed acquisite, di psicopatia. *Riv. di Patol. nerv. e ment.*, Vol. x., f. 10, 1905, p. 449.
- LUNDQUIST. Nagra nyare bidrag till kannedomen om nervfibriller, samt därmed förknippade förhållanden. *Upsala Läkare. Förhand.*, Nov. 23, 1905, p. 86.
- LUGARO. Zur Frage der autogenen Regeneration der Nervenfasern. *Neurol. Centralbl.*, Dec. 16, 1905, S. 1143.
- CLARENCE LOEB. Some Cellular Changes in the Primary Optic Vesicles of Necturus. *Journ. Comp. Neurol. and Psychol.*, Nov. 1905, p. 459.
- DIAS DE BARROS. Contribuição ao estudo da atrophia varicosa das dendrites das cellulas nervosas medullares do porco domestico sob a acção da peçonha da jararaca preguiçosa. *Arch. Brasil. de Psychiat. e Neurol.*, Anno i., No. 3-4, 1905, p. 261.
- LACHE. Sur le nucléole de la cellule nerveuse—Morphologie. *Journ. de Neurol.*, nov. 20, 1905, p. 501.
- HOWARD. The relation of lesions of the Gasserian and Posterior Root Ganglia to Herpes. *Am. Journ. of Med. Sci.*, Dec. 1905, p. 1012.

### CLINICAL NEUROLOGY AND PSYCHIATRY

#### GENERAL—

- ROXO. Noções geraes sobre o systhema nervoso. *Arch. Brasil. de Psychiat. e Neurol.*, Anno i., N. 3-4, 1905, p. 289.

**MUSCLES—**

- LEIR et SONNIOT. Myoclonotonie acquise. *Rev. d'hygiène et de méd. infant.*, No. 5, 1905, p. 569.  
 MEARA. Myotonia Congenita, or Thomsen's Disease. A case. *Arch. of Pediat.*, Nov. 1905, p. 812.  
 HECHT. Myoclonus Multiplex. *Amer. Journ. of Med. Sci.*, Dec. 1905, p. 1041.  
 LECLERC et SARVONAT. Un nouveau cas de Myasthenia gravis. *Rev. de méd.*, Nov. 1905, p. 862.

**PERIPHERAL NERVES—**

- LEREMBOURE. Plaies du Nerf Radial au tiers supérieur de l'Avant-bras. *Thèse de Paris*, 1905.  
 ACCHIOTÉ. Un cas de Névrite du Radial. (Soc. de Neurol.) *Rev. Neurol.*, nov. 30, 1905, p. 1123.  
 ESPOSITO. Amiotrofia da trauma nervoso periferico. *Il Manicomio*, Anno xxi., No. 2, 1905, p. 254.  
 BUMKE. Ueber die secundären Degenerationen nach Verletzung der ersten Halswurzel beim Menschen. *Neurol. Centralbl.*, Dec. 16, 1905, S. 1138.  
 LORTAT-JACOB et SABAREANU. Les sciatiques radiculaires. *Rev. de Méd.*, nov. 1905, p. 917.

**SPINAL CORD—**

- FELIX ROSE. Du tonus et des réflexes dans les sections et compressions supérieures de la moelle. Roussel, Paris, 1905.  
 Tabes.—CLAPHAM. A Note on Tabes Dorsalis. *Journ. Roy. Army Med. Corps*, Dec. 1905, p. 673.  
 EDGEWORTH. On some anomalous cases of Locomotor Ataxy. *Bristol Med.-Chir. Journ.*, Sept. 1905, p. 235.  
 NEUMANN. Hämatemeses bei organischen Nervenerkrankungen (Tabes). *Deutsche Ztschr. f. Nervenheilk.*, Bd. 29, H. 5-6, 1905, S. 398.  
 Friedreich's Ataxia.—LANNOIS et POROT. Le cœur dans la maladie de Friedreich. *Rev. de Méd.*, Nov. 1905, p. 853.  
 Poliomyelitis Anterior Acuta.—GOROVITZ. Du Type Radiculaire supérieur de l'Atrophie Musculaire dans la Poliomyélite aiguë de l'Enfance. *Thèse de Paris*, 1905.  
 MORVAN. Contribution à l'étude de la Paralysie Spinale antérieure aiguë de l'adulte. *Thèse de Paris*, 1905.  
 FLATAU. Die Poliomyelitis anterior acuta. Koenig, Leipzig, 1906, M. 1.  
 Chronic Anterior Poliomyelitis.—MOLEEN AND SPILLER. Chronic Anterior Poliomyelitis. *Am. Journ. of Med. Sci.*, Dec. 1905, p. 1025.  
 Syringomyelia.—KETCHEN. Notes on a Case of Syringomyelia. *Lancet*, Dec. 9, 1905, p. 1685.  
 WEISENBURG AND THORINGTON. A Case of Syringomyelia, with Double Optic Neuritis. *Am. Journ. of Med. Sci.*, Dec. 1905, p. 1019.  
 Paraplegia.—LEJONNE et LHERMITTE. Les paraplégies d'origine lacunaire et d'origine myélopathique chez les vieillards. *Arch. gén. de méd.*, nov. 28, 1905, p. 3009.  
 Syphilitic Paralysis.—NONNE. Ein weiterer Beitrag zur Lehre von der anatomischen Grundlage der "Syphilitischen Spinalparalyse." *Deutsche Ztschr. f. Nervenheilk.*, Bd. 29, H. 5-6, 1905, S. 869.  
 Spine.—W. E. MAW. A Case of Fracture of the First Cervical Vertebra (the Atlas). *Lancet*, Nov. 25, 1905, p. 1544.

**BRAIN—**

- TROLARD. Au sujet de l'avant-mur. *Rev. Neurol.*, nov. 30, 1905, p. 1068.  
 SPANBOCK. Über die Erregbarkeitsschwankungen der motorischen Gehirnzentren und über den Wechsel der Reizeffekte von der Gehirnrinde aus unter dem Einfluss verschiedener Agentien. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 29, H. 5-6, 1905, S. 431.  
 Meningitis.—MAYER. Über traumatische Meningitis. Speyer und Kaerner, Freiburg, 1905, M.—80.  
 Hemiplegia.—SOCA. Sobre la hemiplegia dolorosa de origen central. *Arch. Brasil. de Psychiat. e Neurol.*, Anno i., N. 3-4, 1905, p. 225.

- ERNEST FREY. Beiträge zur Lehre der posthemiplegischen Bewegungsstörungen. *Neurol. Centralbl.*, Dec. 1, 1905, S. 1104.
- EDUARD MÜLLER. Ueber das Verhalten der Blasenenthätigkeit bei cerebraler Hemiplegie. *Neurol. Centralbl.*, Dec. 1, 1905, S. 1101.
- LEWANDOWSKY. Über die Bewegungsstörungen der infantilen cerebralen Hemiplegie und über die Athétose double. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 29, H. 5-6, 1905, S. 339.
- Tumeur.**—PEREIRA. Cysticercose humana. *Arch. Brasil. de Psychiat. e Neurol.*, Anno i, N. 3-4, 1905, p. 317.
- GARBINI. Tumore della fossa posteriore del cranio. *Il Manicomio*, Anno xxi., No. 2, 1905, p. 189.
- ESPOSITO. Psammomi della dura madre. Pseudo-porencefalia e paresi spastica. *Il Manicomio*, Anno xxi., No. 2, 1905, p. 129.
- GROSS. Kasuistischer Beitrag zur Differentialdiagnose des Tumor cerebri und des chronischen Hydrocephalus. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 29, H. 5-6, 1905, S. 456.
- KRON. Ein klinischer Beitrag zur Lehre der sogenannten Akustikustumoren. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 29, H. 5-6, 1905, S. 450.
- JAMES TAYLOR. A Lecture on Intracranial Tumours. *Brit. Med. Journ.*, Dec. 2, 1905, p. 1437.
- Abscess.**—NIESSL v. MAYENDORF. Ein Abszess im linken Schläfenlappen. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 29, H. 5-6, 1905, S. 383.
- Injury.**—LOUIS MOREL. Trois interventions d'urgence pour fractures du crâne avec symptômes de localisation. *Arch. gén. de méd.*, nov. 21, 1905, p. 2949.
- General Paralysis.**—MARIE et PELLETIER. Le mal perforant dans la paralysie générale. *Rev. de Psychiat.*, nov. 1905, p. 469.
- Cerebellum.**—E. W. ROUGHTON. A Clinical Lecture on a Case of Cerebellar Abscess due to Infection through the Internal Auditory Meatus, *Lancet*, Dec. 2, 1905, p. 1597.
- LÉRI et LABRÉ. Forme anormale de Maladie Familiale (Hérédo-ataxie cérébelleuse?). (Soc. de Neurol.) *Rev. Neurol.*, nov. 30, 1905, p. 1105.

#### MENTAL DISEASES—

- FAUSER. Zur allgemein Psychopathologie der Zwangsvorstellungen und verwandter Symptome. *Centralbl. f. Nervenheilk. u. Psychiat.*, Dec. 15, 1905, S. 933.
- FAUSER. Zur Psychol. des Symptoms der rhythmischen Betonung bei Geisteskranken. *Zeitschr. f. Psychiat.*, Nov. 1905, p. 637.
- TEGTMEYER. Korsakow'sche Psychose mit weitgehender Besserung der schweren polyneuritischen Erscheinungen. *Zeitschr. f. Psychiat.*, Nov. 1905, p. 737.
- JENDRASSIK. Ueber die Entstehen der Hallucination und des Wahnes. *Neurol. Centralbl.*, Dec. 1, 1905, S. 1089.
- FRANCO DA ROCHA. Sobre a psychose maniaco-depressiva. *Arch. Brasil. de Psychiat. e Neurol.*, Anno i, N. 3-4, 1905, p. 279.
- THALBITZER. Melancholie und Depression. *Zeitschr. f. Psychiat.*, Nov. 1905, p. 775.
- BENON. Les troubles psychiques chez les hémiplegiques organiques internés. *Thèse*. Roussel, Paris, 1905.
- D'ORSAY HECHT. A Study of Dementia Praecox. *Journ. Nerv. and Ment. Dis.*, Nov. 1905, p. 689.
- SANDRI. La formola emo-leucocitaria nella demenza precoce. *Riv. di Patolog. nerv. e ment.*, Vol. x., F. 10, 1905, p. 464.
- BOURNEVILLE. Contribution à l'étude de la démence épileptique infantile. *Arch. de Neurol.*, Nov. 1905, p. 338.
- ALBRECHT. Zur Symptomatologie der Dementia Praecox. *Zeitschr. f. Psychiat.*, Nov. 1905, p. 659.
- LOMER. Das Verhältnis der Involutionspsychosen zur juvenilen Demenz. *Zeitschr. f. Psychiat.*, Nov. 1905, p. 769.
- DROMARD. Considérations pathogénique sur le mutisme et la sitiphobie des déments précoces. *Ann. Medico-Psychol.*, No. 3, 1905, p. 374.
- ROSENFELD. Ueber Partialdefekte bei Endzuständen der Katatonie. *Centralbl. f. Nervenheilk. u. Psychiat.*, Dez. 1, 1905, S. 893.
- KNAPP. Die polyneuritischen Psychosen. Bergmann, Wiesbaden, 1905, M. 4.
- REICHARDT. Über die Bestimmung der Schädelkapazität an der Leiche. *Zeitschr. f. Psychiat.*, Nov. 1905, p. 787.
- GOTTGÉTREU. Beitrag zur Klinik der Kinderpsychosen. *Zeitschr. f. Psychiat.*, Nov. 1905, p. 759.

- TATY et CHAUMIER. Evolution des états hypochondriaques. *Journ. de Neurol.*, Déc. 5, 1905, p. 521.  
 SIDNEY SCHWAB. Psychasthenia: Its Clinical Entity illustrated by a Case. *Journ. Nerv. and Ment. Dis.*, Nov. 1905, p. 721.  
 SHERLOCK. Lunacy Practice in Germany. *Lancet*, Nov. 25, 1905, p. 1565.  
 TOMASIMI. Le psicosi nell'esercito e la istituzione di manicomi da campo. *Il Manicomio*, Anno xxi., No. 2, 1905, p. 173.  
 URQUHART. Observations on the Heredity of Insanity. *Brit. Med. Journ.*, Dec. 16, 1905, p. 1571.  
 ROBERT JONES. Prognosis in Mental Diseases. *Brit. Med. Journ.*, Dec. 16, 1905, p. 1578.  
 KRAPELIN. Loucura dos querelantes. *Arch. Brasil. de Psychiat. e Neurol.*, Anno i., N. 3-4, 1905, p. 301.  
 MOREIRO e PEIXOTO. Classificação de molestias mentaes do Prof. E. Krepelin. *Arch. Brasil. de Psychiat. e Neurol.*, Anno i., N. 3-4, 1905, p. 310.  
 R. RÉGIS. Précis de Psychiatrie. Octave Doin, Paris, 1905, 10 fr.

#### GENERAL AND FUNCTIONAL DISEASES—

- Chorea.**—KING. A Study of Chorea Minor. *Canad. Practit.*, Dec. 1905, p. 674.  
**Epilepsy.**—ROCH. Des crises épileptiformes d'origine pleurale. *Rev. de Méd.*, Nov. 1905, p. 884.  
 NÄCKE. Die Spätepillepsie im Verlaufe chronischer Psychosen. *Zeitschr. f. Psychiat.*, Nov. 1905, p. 695.  
 EDWIN BRAMWELL and T. GRAHAM BROWN. Observations on Brown-Séquard's Epilepsy. *Rev. Neurol. and Psychiat.*, Dec. 1905, p. 776.  
 HASKOVEC. Luxation habituelle de l'épaule dans les cas d'épilepsie avec antécédents syphilitiques. (Soc. de Neurol.) *Rev. Neurol.*, nov. 30, 1905, p. 1122.  
 THOMAS et NORRERO. Épilepsie Jacksonienne chez un enfant atteint d'hémiplégie légère avec hémiatrophie. (Soc. de Neurol.) *Rev. Neurol.*, nov. 30, 1905, p. 1095.  
**Hysteria.**—BRUNS. Die Hysterie im Kindesalter. Marhold, Halle, 1906, M. 1.80.  
 VICTOR DE BRITTO. A proposito de um caso de coxalgia hysterica. *Arch. Brasil. de Psychiat. e Neurol.*, Anno i., N. 3-4, 1905, p. 295.  
 KOLLARITS. Torticollis mentalis (hystericus). *Deutsche Ztschr. f. Nervenheilk.*, Bd. 29, 1905, S. 413.  
 LÉON TIXIER. Quelques considérations sur un cas d'aphasie hystérique consécutif à un traumatisme important de la région rolandique gauche. *Arch. gén. de méd.*, nov. 28, 1905, p. 3028.  
**Neurasthenia.**—STERN. Ueber sexuelle Neurasthenie. Leipzig, 1905, M. 1.50.  
**Exophthalmic Goitre.**—CROUZON. Un cas de maladie de Basedow traité par le Sérum de mouton Éthyroïdé. (Soc. de Neurol.) *Rev. Neurol.*, nov. 30, 1905, p. 1118.  
 LAIGNEL-LAVASTINE et THAON. Syndrome de Basedow chez une Goitreuse avec Trophœdème. (Soc. de Neurol.) *Rev. Neurol.*, nov. 30, 1905, p. 1106.  
**Rheumatism.**—POYNTON and PAINE. Some Investigations of the Nervous Manifestations of Acute Rheumatism. *Lancet*, Dec. 16, 1905, p. 1760.  
 GAUCKLER et RIEDER. Un cas de Rhumatisme Chronique pouvant servir de type de transition entre le rhumatisme chronique proprement dit et les Arthropathies nerveuses. (Soc. de Neurol.) *Rev. Neurol.*, nov. 30, 1905, p. 1109.  
**Scleroma.**—RAHR. Ein Fall von Sclerom bei einem 6-jährigen Mädchen. *Archiv. f. Kinderheilk.*, B. 42, H. 3-4, p. 258.

#### ALCOHOLISM, ETC.—

- ARONADE. Die Alkoholpsychosen in der psychiatrischen Klinik zu Freiburg, 1887-1905. Speyer und Kaerner, Freiburg, 1905, M. —80.

#### SPECIAL SENSES AND CRANIAL NERVES—

- HOLDEN. The Early Ocular Signs of Dementia Paralytica. *Journ. Nerv. and Ment. Dis.*, Nov. 1905, p. 713.  
 ALEXANDER DUANE. Paralysis of Divergence. *Ophthalmology*, Vol. ii., No. 1, Oct. 1905.  
 ZENTMAYER. Paralysis of the Upward Movement of both Eyes. *Ophthalmology*, Vol. ii., No. 1, Oct. 1905.  
 ARCHIBALD PERCIVAL. The Diagnosis of Ocular Paralysis. *Lancet*, Dec. 2, 1905, p. 1612.



HÜBNER. Ueber die psychische und sensible Reaktion der Pupillen. *Centralbl. f. Nervenheilk. u. Psychiat.*, Dec. 15, 1905, S. 945.

GARHAMMER. Thrombose im Gebiete der Vena centralis retinae. Speyer und Kaerner, Freiburg, 1905, M. 1.20.

STEPHENSON. On some of the Diseases and Injuries of the Eye peculiar to Children. *Brit. Journ. of Child. Dis.*, Dec. 1905, p. 540.

BABINSKI. Hyperexcitabilité électrique du Nerf Facial dans la Paralyse Faciale. (Soc. de Neurol.) *Rev. Neurol.*, nov. 30, 1905, p. 1098.

#### MISCELLANEOUS SYMPTOMS, ETC.—

FRENKEL-HEIDEN. Zur Kenntnis der Psychosen nach Erysipel. *Monatsschr. f. Psychiat. u. Neurol.*, Nov. 1905, p. 383.

THOMAS et LEENHARDT. Un cas de Commotion Médullaire avec paralysie des membres supérieurs et intégrité absolue des membres inférieurs. (Soc. de Neurol.) *Rev. Neurol.*, nov. 30, 1905, p. 1102.

SAXL. Das "Streckphänomen," ein Beitrag zur Kenntnis der Mitbewegungen. *Neurol. Centralbl.*, Dec. 16, 1905, S. 1140.

BALLET et TAGUET. Tic inhibitoire du Langage articulé datant de l'enfance. (Soc. de Neurol.) *Rev. Neurol.*, nov. 30, 1905, p. 1101.

GEORGE R. STILL. A Lecture on Habit Spasm in Children. *Lancet*, Dec. 16, 1905, p. 1754.

KIROFF. Le Signe de Babinski dans la Scarlatine. (Soc. de Neurol.) *Rev. Neurol.*, nov. 30, 1905, p. 1119.

SOUQUES et POISOT. Origine périphérique des Hallucinations des Membres Amputés. (Soc. de Neurol.) *Rev. Neurol.*, nov. 30, 1905, p. 1112.

LIEBMANN. Vorlesung über Sprachstörungen. Heft 6. Kinder, die schwer lesen, schreiben und rechnen lernen. Coblentz, Berlin, 1906, M. 2.40.

SCHLESINGER. Über Sensibilitätsstörungen bei akuter lokaler Ischämie. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 29, H. 5-6, 1905, S. 375.

ERB. Zur Kasuistik der intermittierenden angiosklerotischen Bewegungsstörungen (Dysbasie, Dyskinesie) des Menschen. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 29, H. 5-6, 1905, S. 465.

#### TREATMENT\*—

DOUMER et MAES. Un cas de Paralysie agitante très améliorée par le traitement électrique. *Ann. d'Électrobiol. et de Radiol.*, No. 5, 1905, p. 620.

CHRISTIAN. Quelques réflexions sur le traitement des maladies mentales. *Ann. Medico-Psychol.*, No. 3, 1905, p. 408.

HUTCHINSON. The Surgical Treatment of Facial Neuralgia. Bale, Sons and Danielsson, Ltd., London, 1905, 7s. 6d.

SADGER. Die Hydriatik der Psychosen. *Centralbl. f. Nervenheilk. u. Psychiat.*, Nov. 15, 1905, S. 853.

AUFFRET. Transplantations tendineuses dans le traitement de la paralysie infantile du membre inférieur. *Thèse*. Steinheil, Paris, 1905.

BROWNING. Some Useful Principles in the Treatment of Cerebro-Spinal Meningitis. *Pediatrics*, Nov. 1905, p. 702.

\* A number of references to papers on Treatment are included in the Bibliography under the individual Diseases.

# **Review**

of

## **Neurology and Psychiatry**

---

### **Original Articles**

#### **THE PATHOLOGY OF GENERAL PARALYSIS OF THE INSANE**

By **W. FORD ROBERTSON, M.D.**,  
Pathologist to the Scottish Asylums.

(**The Morrison Lectures for 1906.**)

#### **LECTURE I.**

Delivered on 24th January 1906.

I HAVE been led to select the pathology of general paralysis of the insane as the subject of these lectures, chiefly owing to the fact that its elucidation has formed the principal object of research in the Laboratory of the Scottish Asylums during the last three or four years. There are, however, other reasons for which it is fitting that I should on this occasion direct attention to some of the problems connected with this special form of insanity. General paralysis of the insane, dementia paralytica, or progressive paralysis, is a very common and important disease. It appears to be increasing; it is certainly one of the most terrible maladies that can afflict a human being; it is fatal, with rare exceptions, within a few years; and its etiology and pathogenesis, notwithstanding many positive and dogmatic assertions regarding them, have hitherto been a profound mystery.

In 1904, 1795 persons succumbed to this disease in the asylums of England and Scotland. In the same year, 49 out of

a total of 262 admissions to the Royal Edinburgh Asylum, or 10·7 per cent., were cases of this disease. In some English asylums the proportion is even higher. Thus at the Durham County Asylum it reaches to about 16 per cent. On the continent of Europe the disease is even more prevalent than in our own country. For example, at the asylum of Naples, about 30 per cent. of the patients admitted are general paralytics.

The disease has lately been increasing both in this country and abroad. For example, the annual mortality from general paralysis in the Royal Edinburgh Asylum has risen from 25·5 per cent. of the total deaths in the five years 1890-1894, to 31 per cent. in the five years 1901-1905. The total number of deaths from general paralysis in the English and Scottish asylums has risen from 1321 in 1894 to 1795 in 1904.

To show the gravity of the disease, it is sufficient to mention its leading features. These have been summed up by Dr Clouston<sup>1</sup> in a concise clinical definition, as follows:—"An organic disease of the cortical part of the brain, characterised by progression, by the combined presence of mental and motor symptoms, the former always including mental enfeeblement and mental facility and often delusions of grandeur and ideas of morbid expansion or self-satisfaction; the motor deficiencies always including a peculiar defective articulation of words, and always passing through the stages of fibrillar convulsion, inco-ordination, paresis, and paralysis; the diseased process spreading to the whole of the nerve tissues in the body; being as yet incurable, and fatal in a few years."

It may be added that general paralysis is a disease of the rich and the great as well as of the poor. It is by no means confined to the lower social strata. A few years ago a great statesman fell a victim to it, and to-day is the anniversary of his death. Unlike tuberculosis, cancer and many other maladies, it is a disease about which the public understands almost nothing. By them it is merged with other forms of insanity, which, even in this intellectual age, they still look upon as a mysterious and fatal visitation of a nature entirely different from that of disease as they comprehend it. Nevertheless, it may be said that if general paralysis, and the closely allied disease tabes dorsalis, had been unknown, and were then suddenly to make their appear-

<sup>1</sup> *Clinical Lectures on Mental Diseases*, 1904.

ance and to assume the proportions they now attain in this and many other countries, the occurrence would certainly be universally regarded as one of the most appalling calamities that had ever visited the human race.

Now, the subject of the pathology of general paralysis is far too large to be dealt with exhaustively in three lectures, and I shall not attempt so impossible a task. The time I have at my disposal I shall devote mainly to giving an account of the researches recently carried out by my colleagues and myself. I shall refer to controversial questions merely in so far as it is necessary to do so in order to make clear the new position reached as the result of our investigations.

Before proceeding to give an account of these researches, I must briefly indicate the present position of authoritative opinion regarding the pathology of general paralysis. The question chiefly discussed in recent years has been that of the relation of the disease to syphilis. Professor Bianchi<sup>1</sup> has well remarked that three periods may be recognised in the progress of this discussion. In the first period it was maintained that general paralysis is simply a manifestation of syphilis. It was, however, soon found that antisyphilitic remedies have no beneficial effect, and consequently the second period was reached in which it was maintained that the disease is determined, not by the direct action of the syphilitic toxines, but by a secondary auto-intoxication which may follow this action. This was the parasyphilitic or metasyphilitic period. The third period is the present, in which there has arisen a feeling of scepticism as to the parasyphilitic toxines, the existence of which has never been demonstrated, and which are indeed entirely hypothetical. There are still many who hold that the disease is essentially syphilitic in its origin, but probably most writers on the subject now dissent from this view, and maintain that there are other no less potent factors, such as alcoholism, the excessive use of nitrogenous foods, heredity, etc. "Parasyphilis in the genesis of progressive paralysis," says Professor Bianchi, "is a neologism that harmonises with no proven and demonstrable fact. It is, indeed, the product of a premature induction."

There is similar difference of opinion as to the exact nature of the pathological processes initiated by these supposed etiological

<sup>1</sup> *Annali di Neurologia*, 1902.

factors. Some writers maintain that there is what they term "a premature involution" of the cortical neurons, and that all the other changes are secondary. Others hold that the cerebral vascular lesions are the first to occur, and that the destructive alterations in the nervous tissues follow as a consequence. It is now becoming more and more generally recognised that the cerebral lesions are dependent upon some form of toxæmia.

The pathological anatomy of the disease, as far as it concerns the nervous system, has been minutely studied by hundreds of investigators. The outstanding facts already ascertained are briefly as follows. The cortical nerve cells show acute and chronic degenerative changes, which, like the other cerebral lesions, affect the anterior portion of the brain more severely than the posterior. The medullated nerve fibres of the brain also show more or less extensive degeneration. The tangential fibres are, as a rule, specially involved, though certainly not in all cases. The neuroglia undergoes hypertrophy and proliferation, leading to a condition of cerebral sclerosis. This morbid process, occurring in localised areas in the walls of the ventricles, gives rise to the well-known granulations of the ependyma. The vessels of the brain constantly show chronic or acute irritative changes in their walls, marked by increase of fibrous tissue and proliferation of cellular elements. Special attention has in recent years been directed to the presence of plasma cells in this situation. These are angular cells having a granular protoplasm which stains deeply with methylene blue, but which generally presents a distinct, comparatively clear area. Their presence is said to be almost pathognomonic of general paralysis. The pia-arachnoid is always more or less thickened by inflammatory changes. The spinal cord commonly shows some degenerated fibres, especially in the crossed pyramidal tracts and in the posterior columns. Not infrequently there are well marked tabetic lesions. The cranial and peripheral nerves are often involved in the morbid process. Much attention has lately been directed to the presence of lymphocytes in the cerebro-spinal fluid withdrawn by means of lumbar puncture. Normally this fluid contains very few cell-elements. When the existence of other inflammatory conditions can be excluded, a distinct increase in the number of lymphocytes, or lymphocytosis, is regarded as an important sign of either general paralysis or tabes dorsalis.

I come now to the researches carried out in the Laboratory of the Scottish Asylums and at the Royal Edinburgh Asylum. I shall take them in chronological order, and endeavour to lead you step by step along the road that has been traversed. Before the end is reached, I hope to have laid before you such evidence as will satisfy you that the commonly accepted hypotheses regarding the pathology of general paralysis are erroneous, that, notwithstanding the enormous amount of labour that has already been expended in investigating the disease, the great essential fact in its pathology has hitherto been missed, and that general paralysis is an infective disease, as specific in its causation as tuberculosis, typhoid fever, or diphtheria.

For several years I studied the brains of general paralytics in the orthodox way, and succeeded only in repeating the observations of others. In course of time it became apparent to me that in studying the cerebral changes I was only examining the effects of a toxic action, and that the toxines must have their origin somewhere outside the brain. Further, I was convinced, then as now, that the syphilitic hypothesis does not account for the known facts regarding the disease. I therefore endeavoured to find evidence of the occurrence of a general toxæmia, and to localise the seat of origin of the toxines. About the same time Dr Lewis C. Bruce made independent clinical investigations, having a similar aim. He studied especially the temperature changes, the condition of the blood, the gastro-intestinal disorders, and certain reactions of the blood serum. In a paper<sup>1</sup> published in 1901, he recorded the results of continuous observations made upon the temperature and leucocytes in individual cases. He endorsed Dr Macpherson's opinion that the most characteristic temperature in general paralysis is a recurrent febrile attack every one or two weeks. He also ascertained that leucocytosis and hyper-leucocytosis accompany the rises of temperature, and that in the third stage leucocytosis commonly occurs from time to time without any elevation of temperature. He inferred from these observations that each febrile attack represents the resistive reaction of the body to some toxic substance, and each inter-febrile period an intermission when the resistive powers of the patient have subdued the action of the toxine. He concluded that general paralysis is a disease directly due to poisoning by

<sup>1</sup> *Brit. Med. Journ.*, June 29, 1901.

the toxins of bacteria, whose point of attack is through the gastric and intestinal mucous membrane. There was evidence that the *bacillus coli* is one at least of the organisms concerned in the production of this toxæmia. In a paper<sup>1</sup> published at the same time as that of Dr Bruce, I maintained similar views on the ground of the results of an examination of the pathological changes occurring in the alimentary tract in a series of cases. I found that there was constantly a severe degree of chronic atrophic catarrh affecting the stomach or small intestine, or both, and that the morbid changes appeared to be associated with excessive development of bacteria in the alimentary tract.

Further evidence of the existence of a chronic toxæmia was found in the occurrence of chronic endarteritis in the extra-cerebral vessels. Dr A. Ainslie examined numerous arteries from various parts of the body and found that the condition was constant, though irregular in distribution, and that it was often extremely well marked. About this time another worker in the Laboratory of the Scottish Asylums, Dr Chalmers Watson,<sup>2</sup> advanced very similar views regarding the pathogenesis of tabes, arguing that all we can logically conclude from the fact that a syphilitic history can be traced in a large number of tabetic subjects is that syphilis alters the physiological conditions in such a way as to favour the attack and operation of the actual cause of tabes and allied conditions.

In 1902, Dr Douglas M'Rae, Dr John Jeffrey and I commenced a bacteriological investigation of cases of general paralysis with a view to ascertaining if any facts could be elicited that would throw light upon the nature of the supposed bacterial toxæmia. It may be noted here that five Italian observers had previously made bacteriological investigations in cases of this disease. The blood, the urine, and the cerebro-spinal fluid were examined by one or more of these observers, various micro-organisms being found, but I think it may be said with fairness that no very noteworthy addition to our knowledge of the pathogenesis of general paralysis has resulted from their researches. Dr M'Rae, Dr Jeffrey and I made post-mortem cultures from the inflamed gastro-intestinal tract, the bronchi, lungs, brain, etc. Among the numerous organisms obtained there was one which, from the constancy of its presence in the alimentary or respiratory tract,

<sup>1</sup> *Brit. Med. Journ.*, June 29, 1901.

<sup>2</sup> *Brit. Med. Journ.*, June 1, 1901.

by its occasional occurrence in the brain and in view of the ascertained pathogenic characters of the group to which it appeared to belong, there seemed reason to believe might have special importance. This was an organism resembling the Klebs-Löffler bacillus. Cultures of a bacillus of this nature were obtained from seventeen cases out of twenty examined. In the remaining three a similar organism was afterwards found in sections of the alimentary tract. Cultures were obtained from the brain in four out of seventeen cases. On the grounds of our observations, we advanced the hypothesis that general paralysis is the result of a chronic toxic infection from the respiratory and alimentary tracts, permitted by general and local impairment of the defences against bacteria, and dependent upon the excessive development of various bacterial forms, but especially upon the abundant growth of a Klebs-Löffler bacillus of modified virulence, which gives the disease its special paralytic character.<sup>1</sup> In our later investigations, Dr M'Rae and I have simply been putting this hypothesis to the test, and every step forward has been attended with the elucidation of some fresh fact that has rendered it more probable.

I followed up these bacteriological researches by a histological investigation of the supposed infective foci. In a series of twenty cases of general paralysis I was able to recognise in the catarrhal exudations in the respiratory and alimentary tracts a bacillus identical in form and staining reactions with the organism isolated by cultural methods. In several of the cases it was present in very large numbers. In the course of these histological investigations, a filamentous organism having special characters was observed in five cases, either in the walls of the bronchi or alimentary tract, or of both. I stated that there were some grounds for supposing that this organism is a thread form of the diphtheroid bacillus and that its presence in great numbers in the lymphatics of the respiratory or alimentary tract represents a terminal invasion by this bacillus.<sup>2</sup>

Dr Shennan and I have made two series of experimental observations with a view to ascertaining if these diphtheroid bacilli are capable of producing in lower animals changes in any way resembling those that occur in general paralysis. We used

<sup>1</sup> *Review of Neurology and Psychiatry*, May 1903.

<sup>2</sup> *Review of Neurology and Psychiatry*, July 1903.



chiefly a bacillus isolated from the bronchus of a case in which in this situation there was found to be a very abundant invasion by the filamentous form of the organism. We have made two series of experiments, but an account<sup>1</sup> has as yet been published only of the first series, and I shall confine myself to it.

It was ascertained that the organism was non-pathogenic to guinea-pigs. Intra-pleural injection in a white rat resulted in death of the animal in five days. Microscopical examination of the tissues showed that the organism had multiplied at the seat of injection and had spread to the adjacent pulmonary tissues and also to the pericardium. The invading organism was beginning to assume a thread form. Three rats were fed for several weeks upon bread mixed with unsterilised broth cultures of the bacillus. After three or four weeks they began to show morbid symptoms, which gradually increased in severity until the animals became acutely ill. At first they showed especially slowness and uncertainty of gait and drowsiness. Later they manifested distinct motor weakness, marked inco-ordination of movement, dyspnoea and great drowsiness. One rat was killed with chloroform when it appeared to be moribund. In the other two the disease was allowed to go on to a fatal termination, which occurred about two months from the time of the commencement of the feeding with cultures. Control animals remained healthy. Microscopical examination of the tissues revealed in each animal a similar series of morbid changes. There was well marked catarrh of the alimentary tract in all three, and a similar condition of the bronchi in two, accompanied by some catarrhal pneumonia. The diphtheroid bacillus was found in the catarrhal exudations, but its detection presented the same difficulties as in cases of general paralysis. A large proportion of the nerve cells of the cerebral cortex and spinal cord were markedly degenerated. The neuroglia, especially in the first layer of the cortex, showed slight but distinct proliferative changes. There was distinct increase of the cell-elements in the walls of the cortical vessels, and also proliferation of the mesoglia cells and of the cells of the pia-arachnoid.

In the two rats in the case of which the illness was allowed to go on to a fatal termination, there was extensive invasion by the filamentous organism already referred to. In one animal the

<sup>1</sup> *Review of Neurology and Psychiatry*, April 1903.

threads were found in the lymphatics of the stomach, duodenum, and ileum, as well as in the liver and in the walls of the bronchi. In the last named situation this invasion exactly reproduced the histological picture to be observed in the case of general paralysis from which the bacillus was isolated. In the other rat this filamentous organism was found in the walls of the stomach, duodenum, and ileum, and also in the capsule of the spleen and in a lymphatic gland. Beyond question these animals present evidence of the occurrence of many of the morbid processes that can be recognised in the nervous system of the general paralytic, but they survived too short a time to make it possible for the complete histological picture to be developed.

At this stage of the investigation, I summarised the case for the diphtheroid hypothesis of the etiology of general paralysis in opening a discussion on the pathology of the disease at the annual meeting of the British Medical Association, held at Swansea in 1903.<sup>1</sup>

An interesting experimental observation has also been made by Dr Lewis C. Bruce. He used cultures derived from the bacillus that was employed by Dr Shennan and myself in our experiments upon the rats. From time to time in the course of several months Dr Bruce injected a goat subcutaneously with these cultures for the purpose of obtaining an immune serum for therapeutic use. After a time the animal developed signs of alimentary disturbances. It had been known to lick the spots at which the injections were made, and probably in this way its alimentary tract became infected with the bacillus. The animal became tottering in its gait, and about six months from the time when the last subcutaneous injection had been made, it had a seizure closely resembling the congestive attack of a general paralytic. It rallied to some extent, but died a few days later. A culture was made from the oesophagus after death, and a growth of a diphtheroid bacillus was readily obtained. Dr Bruce kindly sent me the brain and some of the other organs for examination. The brain shows proliferative changes in the vessel walls, proliferation of the neuroglia and degeneration of the nerve cells, but each of these morbid alterations is slight in degree. Nevertheless, among the proliferating cell-elements in the vessel walls, several distinct plasma cells have been detected.

<sup>1</sup> *Brit. Med. Journ.*, October 24, 1903.

I think it is certain that in this case the part of the nervous system chiefly affected was the spinal cord, which unfortunately was not obtained. The condition of the brain indeed corresponds exactly to that of a case of tabes dorsalis in which mental symptoms have been absent or only very slight.

In May of last year, Dr M'Rae and I reported the results of an investigation in which it was sought to ascertain if diphtheroid bacilli are commonly present in the genito-urinary tract in cases of general paralysis. We have since extended these observations. We have ascertained that female general paralytics constantly suffer from chronic leucorrhœa, and that the discharge always contains abundant diphtheroid bacilli. In three instances the first cultures made from the discharge have yielded a diphtheroid bacillus alone. Diphtheroid bacilli are also constantly present in the urethra of the male general paralytic. Similar organisms have also been found, but generally only in comparatively small numbers, in the same situations in a considerable proportion of cases in which there was no ground for suspecting that the patient was suffering from general paralysis. In seven consecutive cases of general paralysis combined with tabes, we have found the urine to be loaded with diphtheroid bacilli. We have now obtained a culture of a diphtheroid bacillus from the brain in nine cases of general paralysis out of twenty-three from which cultures have been made from this organ. We have also examined the cerebro-spinal fluid removed by lumbar puncture from five cases of general paralysis. In the centrifuge deposit, in addition to lymphocytes, there was always a considerable amount of granular debris, and among this debris, or within the lymphocytes, we have observed in three cases bacilli, which have very little affinity for staining reagents, but which, nevertheless, can not infrequently be recognised to have the morphological characters of diphtheroid bacilli. We have also examined blood films, staining them by methods suitable for the detection of diphtheroid bacilli. We have obtained the blood by a method, already described, which reduces to a minimum the risk of contamination from the skin of the patient. In a preparation from one paralytic, presenting the signs of a slight congestive attack, we have observed a small group of typical diphtheroid bacilli, with distinct metachromatic granules.

Lastly, we have searched for evidence of the presence of diphtheroid bacilli in the walls of the inflamed cerebral vessels, using chiefly various modifications of Neisser's method. No definite results were obtained until last summer, when examining sections of a portion of the brain subjacent to a purulent area in the pia-arachnoid, from which we had obtained a pure culture of a diphtheroid bacillus. Here we found, in a preparation stained by Neisser's method, a small group of faintly coloured, but still quite definitely recognisable diphtheroid bacilli lying in the walls of an inflamed vessel.

The occasional presence of diphtheroid bacilli, generally incapable of taking the stain in the ordinary way, in films of the centrifuge deposit from the cerebro-spinal fluid, in blood films and in sections of the brain, raised the question whether these bacilli were not from time to time gaining access to the blood circulation, and being rapidly destroyed by phagocytic and lysogenic actions. Dr M'Rae and I therefore resolved to study experimentally the action of the phagocytes and blood serum upon diphtheroid bacilli isolated from cases of general paralysis. The flood of light that has been thrown upon the problem of the pathogenesis of general paralysis and of tabes dorsalis by putting this hypothesis to the test, I hope to show in the next lecture.

---

### **A STUDY OF SOME CASES OF DELIRIUM PRODUCED BY DRUGS.**

By DR AUGUST HOCH, Bloomingdale Hospital, White Plains, N.Y.,  
Instructor in Psychiatry, Cornell Medical School, New York.

CASES of the nature of those here recorded are probably not very rare. Nevertheless, during my ten years' service at the M'Lean Hospital, Waverley, Mass., I have had occasion to observe only eight, four of which are here presented. But it seemed to me of some value to establish clearly the delirious nature of these conditions, to analyse them carefully, and to compare them with the deliria about which we are best informed, those produced by alcohol. The excellent monograph by

Bonhoeffer,<sup>1</sup> a model of clinical analysis, has greatly advanced our knowledge of delirium tremens and of deliria in general. The desire was very natural, therefore, to study deliria with a different etiology in a similar manner. That the writer feels a great obligation to Bonhoeffer, whose work in part guided his studies and his conclusions, he desires to express at the outset.

The drugs to which these deliria were attributed are chiefly bromides, hyoscine, various true hypnotics, and morphia, and it is a notable fact that it seems to be of very little consequence which drug is used; indeed, I have seen one case where antifebrin seemed to have been the only, or at any rate, the most important drug. After all, as is assumed in the case of alcohol, the action of the poison introduced is probably only the indirect cause; nor does it seem to be the only one, for insufficient food, protracted loss of sleep, digestive disorders, and general exhaustion, seem to act as contributory causes. We may infer this from the fact that such factors are often present, and that we find occasionally conditions resembling delirious reactions in manic state, for example, after just such causes have been at work. Unfortunately it has mostly been impossible to determine the exact amounts of the drugs taken, and in one case the doses admitted seemed too small to account for the profound reactions. Nevertheless, the experience with all such cases cannot leave any doubt regarding the importance of drugs as an etiological factor in them.

CASE I. Mrs H., aged 51. In the hospital from March 5 to March 28, 1903. The patient had one sister who had the opium habit. Any other neuropathic traits in the family were denied.

The patient had never been insane, but since the age of 30 had complained of very severe headaches which occurred at menstruation, and for years had been in the habit of taking morphia for them to the extent of  $\frac{1}{8}$  to  $\frac{1}{2}$  grain a day. She is said to have been perfectly well in the intervals. For three months before admission the patient had not menstruated, after the flow had been scanty for about a year. Two months before

<sup>1</sup> Bonhoeffer : "Die Geistesstörungen der Gewohnheitstrinker." Gustav Fischer, Jena, 1901.

admission the headaches again came on, and now became continuous; she took morphia, rising rapidly to a grain a day, but, it is said, no farther. This was continued until admission, while in the meantime bromides were added. These, it was claimed, were not in large doses. The patient had become irritable, and two weeks before admission she began to get restless, somewhat apprehensive, and for five or six days before admission she is said to have been confused and at times dull. For a week she had not slept and had scarcely taken any food.

On admission the patient appeared restless, evidently heard voices, but she showed no fear. She was disoriented and used wrong words. At the morning visit on the following day she was found with a rather pasty complexion, a heavily coated tongue, a temp. of  $99.2^{\circ}$ . Her breath was foul. There was no eruption on the body. There was no evidence of any palsies; the movements of the arms were not ataxic, but the gait was rather staggering. There was a general coarse tremor in the hands. The reflexes were of normal intensity. She lay in bed tossing about rather restlessly. Her mood was one of a whining depression, with some irritability, but no apprehensiveness. She looked somewhat dull, and her attention could at times be attracted only with marked difficulty, again quite readily; but we were struck with the fact that now and then, even at the time when we had difficulty in obtaining answers, she made occasional comments on things which were said in her hearing. Hallucinations were at times quite prominent; she had spoken of hearing bells ringing, had seen pictures on the door, her sister in the pillow, a man in her bed, and she tried to pick imaginary flies from the bed-clothes. She was completely disoriented; though she repeatedly called the physician "doctor," the nurse "nurse," yet again she miscalled them. Paraphasic turns of speech were quite marked, as we shall presently show. For the two succeeding weeks her condition remained essentially unchanged, and may be summarised as follows. Sometimes she appeared dull, even to the extent of soiling herself. Her attention varied: it either could be easily attracted or this was very difficult, and she could be pricked with a pin without any reaction. Her disorientation remained, though shaded off gradually. She thought she was in New York and other places; again, called people by wrong names. Her time orientation was very poor.

Sometimes she related delirious experiences ; for example, said that she had been up the river lately in a boat, or that she had just been in the woods, where "some money was tied to a tree," and the like. The hallucinations continued, and even became more marked. She heard voices, reached out her hands to fancied visitors, talked to the wall, spoke of the girls upstairs "who have talked" about her, and quite marked was the fact that she picked up imaginary threads from the bed-clothes. Artificial hallucinations could be produced by rubbing her eyes. On such occasions she said that she saw "a fire-place," "wood-work," "shelves," "a woman in a blue dress," "all sorts of things." Reading tests showed fair results at times ; again, she made glaring mistakes, such as reading "pollies" instead of "1903." When questioned about events in her life she varied a good deal, sometimes gave perfectly absurd answers, *e.g.* that she was born in '81 ; again, the answers were apparently perfectly correct. A few tests to study her ability to retain impressions (*Merkfähigkeit*) yielded results which would make one think that this was very poor ; but the question of attention was not sufficiently considered at the time, so that we must not lay too much stress on the results, all the more so since it was found repeatedly that at the end of an examination she remembered incidents which had taken place at the beginning of it. Her talk may be illustrated by the following examples. She said spontaneously, "I'll never see my mother any more ; she has been trying to hold up since she was lost." And then, pointing to the nurse, she said, "This is my mother. Please let me go. There is nothing for me to stay here. That's what I was, freezing. It seems just like she came in the window." (What do you mean ?) "Well, don't you know there is a store in front of the bridge that comes right down to a point of lace. She lived there, or she did when I lived there," etc. "Down to a point of lace" is evidently a paraphasic turn, a trait which may be further illustrated by the following samples. "We were coming down the ref road . . . I can't tell you where it is, it's the mostly jardmar, in the mell, mell jar, in the worsted mill yard." Or in speaking of Chattanooga, she said, "Chattanulgo, Challamutta" ; and on one occasion when she heard a telephone ringing, she said it was the Chattanooga ringing, or "You are the gentleman I not in the grocery store."

In general it may be said, as is the case in these patients, that though the talk showed some shifting of subjects, loosely connected, it was not that which made it difficult to follow it; nor was this a very marked trait, as she kept often to the subject she had chosen fairly well; but it was the fact that she told of delirious experiences which we knew nothing about, and the talk was further obscured by the paraphasia.

After the two weeks the patient gradually became perfectly clear, orientation was excellent, the hallucinations disappeared, and she talked very naturally. It was all the more striking that with this clearness she retained for a number of days a belief in some of her delirious experiences. This was especially striking since these were so absurd. Thus she claimed that the nurse had told her that she had killed a man, and said she knew it was her husband. She explained that at home her husband discharged a nurse, and that the latter followed him to the barber shop and shot him through the thumb. When questioned retrospectively about the events which had occurred in the hospital, it was found that the very first part was practically a blank to her, but that after that she remembered quite a number of things, which, however, were not put together in anything like a sequence. She was taken home before she had entirely ceased to believe in some of her delirious experiences, although she did not at all react to them.

CASE II. Mrs W., aged 30. In the hospital from May 23 to August 3, 1903. Her maternal grandmother was insane for fifteen years until her death at the age of 60; her mother had repeated attacks of "nervous prostration," and one of the mother's brothers was an epileptic. A paternal uncle had an attack of insanity.

The patient herself had "nervous prostration" when 22, a condition in which she complained of considerable physical weakness, also of much pain in head and spine, and is said to have been very "hypochondriacal." She was in bed for months. She was married two years after the onset, but only two years later, *i.e.* four from the beginning, was she considered really well.

Three months before admission the patient is said to have had an attack of "grippe." She was weak after it, complained of palpitation, and was considerably worried about it. She had to



remain in bed, became nervous and irritable, and more and more worried about her condition. It is claimed that she would sometimes stare for half an hour at a time. A month before admission she attempted suicide for the first time, and was henceforth very insistent in her attempts. Three weeks before admission there were occasional spells of mental clouding, and for a week before admission she had been rambling, noisy, resistive ; finally confused, untidy, hallucinating, eating almost nothing for some days.

Fortunately we have a good account of the drugs which this patient received. It must be remembered that she was admitted on the 23rd of May. From April 1 to 11 she was given 10 grains of bromide at night. From the 11th until the 27th it was replaced by 18 grains of trional, repeated if necessary. From April 26 to May 3 she had 60 drops of Tr. hyoscyamus a day. From May 3 until admission she had regularly, at first 60, then 120 grains of bromide, plus 15 drops of Tr. gelsemium a day. In addition to that she had, for the week preceding admission, altogether  $2\frac{1}{2}$  grains of morphia and  $\frac{4}{100}$  of hyoscine. And finally she was given Tr. passiflora, 5 to 10 drops, every 2 to 3 hours ; later, 30 drops at longer intervals.

The patient was admitted with a temperature of  $100^{\circ}$ , sallow appearance, foul breath, heavily coated tongue, pulse 100. She was restless, shouted for her husband, spoke of hearing her people murdered, of seeing coffins, men with revolvers. She frequently seemed to pick up things from the bedclothes, and when questioned said she saw bugs and threads. Her voice was thick and her talk fragmentary. She was completely disoriented. At the morning visit her physical state was the same as described. In addition it was found that there was no tremor, but marked exaggeration of reflexes, with pronounced ankle clonus, inexhaustible on the right side, exhausted after 10 to 12 motions on the left. Babinsky absent. She lay in bed quietly, mumbling something to herself, occasionally calling out, evidently in response to hallucinations, sometimes picking imaginary things from the bedclothes. She appeared dull. The mood was indifferent, there was neither fear nor any evident depression or exhilaration. It was sometimes very difficult to attract her attention, again more easy. Sometimes she commented on slight, quite unobtrusive noises, such as a distant train. Orientation was poor. She said she did not know where she was, did not know the

people, but she gave the month as May, the year as 1903, then 1902. Again, she said she was at her sister's house, but frequently called the doctor "doctor," the nurse "nurse."

Her talk may be illustrated by the following. When asked how long she had been sick, she said, "I have been sick eight or ten weeks—that is if I speak right—now my folks tried to lose me, they were hunting for me." (Did you see them?) "I could not hear a sound, only her [nurse], and she will kill me" (no affect). "They all say I was afraid because I went to a store on Tremont Ave. They would not let me have—well, she would not let me—have anything to do—you remember that [to nurse]—she can't find out. I'm growing hazier and hazier—but this forenoon, well, I'll tell you what she did. I see her object in it now. I hadn't thought of it. I have been moved so often. We have moved around in the daytime—in the night—we have moved all around, I don't know how many things," etc.

What is not brought out in this sample is her paraphasic turns, which, nevertheless, were quite marked. Thus she said, in good connection, "That is all the satisfaction I can get, and I am satisfaction." Or when asked the day, she said, "I don't know, I haven't seen a map for ages. I am just 8.30 May something." Or again, "Are you the gentleman that's marrying this house?" Or, "He make it distinct enough that I would not get well. Distinct, extinct enough, he made it excitement enough," etc.

When asked memory questions she varied, evidently owing to her variation in responsiveness. She gave her age correctly. (Have you a child?) "Yes, three years ago" (correct). (Is the child living?) "No, dead" (incorrect). (How long ago since it died?) "Two years." (What did it die of?) "Still-birth." (What?) "Two years ago the 8th of February." (What happened then?) "A boy was born to me." (How long did he live?) "Oh. I was taken sick on the 8th and he was born on the 9th." (Is the boy living now?) "Yes." (What is his name?) She gave it correctly. (Have you ever lost a child?) "No" (correct). (How old is your boy?) "33." (No, your boy?) "3" (correct). Then she was asked, "What is 9 times 15?" She said 19. ( $7 \times 13$ ?) "21." ( $8 \times 9$ ?) "72." ( $16 \times 12$ ?) "72." (What is the capital of the U.S.?) "Boston." (Capital of Maine?) "45." (Capital of Maine?) "Capital of Maine?"

75." (What is 75 ?) "A number." Then she was again asked impressively, and she said correctly, "Augusta."

In addition to the hallucinations above described, artificial hallucinations could be produced by rubbing her eyes. She said she saw "a horse-car on the street," "a post," "a white post," "people and a dog." (What kind of people ?) "Mostly Chinese women." (What colour of dresses ?) "Mostly white dresses. I saw a cap just now—all kinds of things, houses and everything else." When told to open the eyes she said, "Now I see a bunch of grapes." Asked what she saw on the ceiling, she said, "Grapes—single grapes, small and large ones."

Just as we found in the other cases, this woman made striking mistakes in her reading.

This condition lasted about 10 days, while the more marked symptoms gradually faded, the tongue became clean, the reflexes normal, the orientation became much better, the paraphasia was slight, the talk was much clearer, but in spite of all this improvement she continued to believe in the delirious experiences and for a time hallucinations persisted. Although she finally cleared up altogether, she held on to some delirious experiences almost to the end, while at the same time she showed a certain mental sluggishness.

CASE III. Mrs E., age, 43. In the hospital from July 22 to September 15, 1904. Heredity is denied, and the patient has never before been insane. A year before admission she had a good deal of worry. She lost flesh and got weak, slept poorly, and it is stated that at that time she took a considerable amount of morphia, but that she had not taken any for three months. For about three weeks she has felt very exhausted, slept poorly, complained of many pains, and it is stated that a great many drugs were then given her, but we were unable to find out just what. She got steadily worse, finally somewhat confused, and three weeks before admission she was sent to Boston. There she had to be looked after, had to be dressed, fed, and gradually became excited and at times fearful, confused, so that 12 days before admission she was taken to a small hospital, where she was dull, untidy, restless, had hallucinations of hearing and vision.

In this hospital she was again given hypnotics, but as has often been our experience, the guilty physicians are very apt to

be exceedingly general in their answers to letters of inquiry about drugs.

The patient was brought to us in a state of marked dulness and hebetude; she showed a tendency to keep her eyes closed, was untidy, her mouth was dry, presented sordes, the tongue was heavily coated, the breath foul, the pulse rather weak (100). The internal organs presented no abnormality. The reflexes were normal. There was no terror. She lay muttering, speaking indistinctly and thickly, but when her attention was attracted her talk was much more connected and the voice much less thick. Sometimes it was easy to attract her attention; again, difficult. But it was quite striking that she repeatedly caught up statements made within hearing and commented on them. Her train of thought was at times difficult to follow, partly on account of paraphasic utterances, partly because she spoke of things irrelevant to the situation. But she kept on the chosen subject remarkably well. The answers were often quite irrelevant, evidently because she either paid no attention to the question or because of her paraphasic turns. We may give a few examples. When asked what is two times two, she said "two over"; and again, asked what's two times two, 'that what I said, you would think I was crazy, a woman of 75 to make me marry' (she had spoken of that before), "to be asked why I did not marry such a woman" (paraphasia). Then turning to the nurse: "Florence. No, that isn't Florence. I said 'put that feather over there,' and Florence said, 'No, I won't put that feather over,'" etc.

The data of her life were at times given well, again poorly. She was totally disoriented, miscalculated people. Even simple multiplications were done poorly. Her mood was either indifferent or somewhat euphoric. Hallucinations were present and frequent, especially those of hearing, and to a lesser degree those of sight. Quite striking were the tactile hallucinations, or tactile and visual combined, which were manifested by her imaginary picking up objects. Her ability to retain impressions tested in the ordinary way (given a number of 4 digits to remember) appeared poor, but here again we must add that such a test is only of value if the mental responsiveness is taken into account. Paraphasia was pronounced. Interesting were the results when objects were shown to her. They were evidently in part due to a disorder in apprehension, so clearly brought out in

Case IV., and quite striking was also the influence perseveration. The following samples may be mentioned. (Knife.) "Brick house." (Knife.) "Those are—" (thinking). (Tell me.) "Knife." (Bunch of keys.) "A key ringer—ringer for keys." (Watch.) "Keys." (Charm.) "A charm." (Spectacles.) "Those rings which go on." (Cuff.) "Keys, cuff of keys with a key-note in it." (Pink.) "Pink." (Palm leaf fan.) "Fan." (Brown book.) "Bible." (Hand-glass.) "Looking-glass." (Comb.) "I don't know—that's my black comb." (Hair-brush.) "Comb. It's a hair brush." (Closed fan.) "A fan, a parasol, a very little parasol." (Opened fan.) "A fan, a parasol." (Cuff button.) "A gold ring." (50 cents.) "A quarter." (25 cents.) "A quarter." (50 cents.) "A quarter." (5 cents.) "10 cents." (One cent.) "5 cents."

Three days after entrance the attention was attracted with greater ease, but the paraphasia persisted to a marked degree. She read very poorly. For example, when made to read "bats have proportionately the longest ears and the oddest shaped noses in the whole animal kingdom," she read, "Bates properly continue the largest earnestly and clearly noses of the kind, of the innumerable kind." When she was shown pictures she showed marked abnormalities, pointed out birds where there were none, called a piece of bread in the hand of a little child "a cucumber squash," saw "a lobster claw" on a piece of paper which contained only indistinct marks, not at all suggestive of a lobster claw to a normal person, or she called three lambs "three cans" (paraphasic?). At that time she was still disoriented as to place. In regard to time she knew the month and year, but nothing more. She miscalled persons, but not consistently. She gave no one a correct name, but called the doctor "doctor," the nurse "nurse." The hallucinations continued. She heard voices, and still picked imaginary things from her bed-clothes. In regard to the disorientation, it may be mentioned that she thought she was at home, or in the house of a friend. She repeatedly told of delirious experiences.

In a few more days the hallucinations left, she became perfectly clear and the attention was good, but she still called the place wrongly, still uttered delirious experiences. Thus she told of an accident which had happened in which her mother had been injured, and claimed that the examining physician had been

called in and had operated on the mother at her home. Gradually she cleared up entirely, not only from her delirium, but from the condition which had originally led to the giving of drugs.

CASE IV. Amelia G., aged 39. Dressmaker. Admitted January 11, 1905.

The patient has some psychopathic heredity, and it is said that she was always of a suspicious nature, was easily frightened, and inclined to be quite hypochondriacal in the sense of making a great deal of small ailments. For ten years she complained much of pain in the neck and head, but on the whole was able to do her work.

Six weeks before admission she complained more of the pain, became depressed, despondent, listless; sometimes she was restless.

Five days before admission she became more depressed, self-accusatory, and sat for hours without speaking. Soon after this she began to "talk queerly," said people were dead, that she had killed six little children. She also said that the top of her head was "blown up." She claimed that her mind was gone. At the same time she showed indications of morbid self-reference, thought things which were done had a peculiar meaning, and she fancied that people looked at her. A few days before admission, hallucinations began; she answered voices, and she saw "red devils crawling over the sister's jacket," "a little angel walking round the rim of her drinking cup." She was often seen staring.

For about a week she had eaten very little and had slept very poorly.

Now, this woman had been given liberal doses of bromides in the six weeks preceding her admission to the hospital. We were unfortunately unable to find out the exact doses, but it is said that she was given a teaspoonful every three hours. The fact that at entrance she had marked acneform eruption on her body also supports the supposition that she had been heavily dosed. Besides these bromides, she was given hypodermic injections, the nature of which we could not find out.

At entrance the patient showed, as was stated, an acneform eruption; the tongue had a heavy brown coat; her breath was foul. Her gait was somewhat unsteady, resembling that of cere-

bellar ataxia. But there was no tremor, the reflexes were normal. The pupils could not be tested on account of lack of co-operation. She showed marked tenderness and pain over the joints of the legs, but no swelling. Her urine showed a slight trace of albumin, but nothing pathological otherwise, except a very high specific gravity, 1.041. Temperature normal. Pulse and respiration showed nothing of any consequence.

She wandered aimlessly about, presenting the uncertain movements above described. Her expression was strikingly empty, but not immobile. She made the impression of being absorbed in vague thoughts, and very often she did not answer questions, or what she did say had no bearing on what she had been asked, but was either a vague allusion to the "Blessed Virgin" or the like, or a repetition of something she happened to hear, and the result was the same whether complicated or the most simple questions were asked. But she showed her tongue, and reacted quickly to pin pricks. Quite striking was an aimless resistance, blind in character, yet without an affectful background to it, making rather the impression of a tendency to perseveration, a trait which was later on brought out more clearly. Interesting is the fact that with this there was at times a tendency to catalepsy, and above all a marked, though not consistent, echopraxia, even to tests. It should be noted that in spite of all these traits she at times occupied herself with the physician, fumbling aimlessly about his clothes and the like.

Next day the condition was quite different and remained different for about a week, after which time it very gradually shaded into a typical state of manic depressive retardation, which persisted so long as I observed her. The condition which developed on the second day, and which we shall presently describe, was a delirious state, and for some weeks after the height of it was passed the slight delirious traits persisted, masking the manic-depressive retardation, so that for quite a while the case presented considerable difficulties to the correct interpretation.

During the delirious condition she was at first completely disoriented as to place, persons, time, even the time of the day. Whether this had been so on the first day we were unable to decide. It could now be established, because, in contra-distinc-

tion to her state of the first day, she frequently answered questions, although she had a marked tendency from time to time to get into a similar staring condition as at first, and even to become decidedly drowsy. These variations in her responsiveness were quite marked, so that at times it was impossible to attract her attention. When thus absorbed she did not react to pin pricks, and, at these times also, it was found that she would firmly hold on to anything which she happened to have in her hands, so that it could not be taken away from her except when it was possible by putting some other object in front of her eyes to forcibly attract her attention to that. Again, when looking at anyone, she would follow that person with her eyes when he moved about. All this made the impression of a peculiar fascination and perseveration. During this time she lay in bed, often appearing rather dull. She hallucinated, saw "staggering things with long legs," "a bird" in the physician's hair, "lots of children at the end of the hall," or she saw faces in the transom, and heard voices. But she had evidently no hallucinations of touch. Her talk, which was rather scanty, was, however, clear, and there were only occasional paraphasic turns in it, but these were distinct. She produced, however, a number of delirious experiences. She said she had been "in a dry goods store this morning," that she had gone down a long street, and the like.

The mood during all this time was strikingly indifferent, even when she uttered occasional depressive ideas.

We then made some experiments daily in order to study more closely the hallucinations, the process of apprehension, and her ability to retain recent impressions. In all these experiments the question of mental responsiveness had to be taken into consideration, so that we also made some tests regarding this.<sup>1</sup>

Let us first consider the hallucinations. Like all the other cases, this patient showed marked artificial hallucinations, *i.e.* when the eyes were pressed upon and she was asked what she saw, she said, for example, "a whole pile of black iron rails"; later, "I see a little girl of 13 or 14 holding a doll." (What kind of dress has she on?) "A grey one." "I see a baby carriage." She also said, "I see a man," or again, "It looked

<sup>1</sup> These experiments I made in conjunction with my friend and associate, Dr S. J. Franz, to whom I wish here to extend my thanks for his assistance.



like a yellow suit with brown buttons on it." As will be remembered, it was Liepmann who first showed that such hallucinations could be produced in alcoholic deliria.

When pictures were shown to her the hallucinations were also very marked, just as had been the case in Mrs H. Thus in one picture which she first described quite well, she added, "and there is a man crawling under the fence." In another picture she pointed out a cat in the grass, where there was none. After having described the essentials of a third picture correctly, she added, pointing to rather small, indistinct geese, that they were birds. A small brown chicken she called a squirrel. Finally she saw "a big snake and a big green lizard." (The picture showed a patch of grass.) When she was shown a fourth picture she again described the essentials correctly, but when she came to an indistinct chick she said, "There is something here but I can't see it." Later she saw "bugs running up the shrubbery," and finally "a long green snake." In other words, the patient began invariably by describing the picture correctly. That was at a time when her attention was attracted by a new picture, but soon she began to hallucinate, and as we shall presently point out, she began to see indistinctly, and when one watched her further she was very apt to go off, as it were, *i.e.* to get into a staring state similar to the one described on the first day, or she got distinctly drowsy.

That she does not see well we infer from the fact that she pointed to the chick saying, "I cannot see that." However, this was rather isolated and usually she hallucinated. Some years ago I had occasion to observe a case of Korsakow's psychosis quite early in the course. This man resembled in many ways the patient under consideration. In that case it was very evident that he had periods when his vision was very indistinct. He also hallucinated at times during these periods of indistinctness of vision; more often this was not the case. The Korsakow case differed very markedly from Miss G. by making a much more natural impression, but from time to time he had peculiar short spells in which he seemed to wander, would not respond, and sometimes even his attention could not be attracted for the space of a minute or so. My attention was first called to this condition while I was making a sensory examination. He would answer promptly for a time, then suddenly he could be touched or

pricked without making any response. In order to study this more carefully we applied the following tests. We read to him columns of thirty-two figures each, among which five threes were irregularly distributed. He was asked to tap the table every time he heard a three. He would often allow from one to five threes to pass unnoticed, on one occasion fourteen in four lines, and altogether 14 per cent. When this man was shown series of letters (we used quite large ones) or pictures, he would at times name or describe them very well. At other times he would say, "it's dull," or "it's blurred," or "it's going," or simply, "I can't see it." Although the most frequent result was that his vision became merely blurred, he, at times, hallucinated like Miss G. For example, on one occasion, instead of seeing a letter he said he saw "a procession of the Knights of Pythias." A few times he also had auditory hallucinations in such periods. Questioned about these states he said, "My mind wanders"; or again, "I get forgetful at those times."<sup>1</sup> We see, then, that this patient had short periods during which his "mind wandered." In these, his attention could at times not be attracted; at other times he showed a peculiar visual disorder, and with it a tendency to hallucinations. The analogy with the case of Miss G. is obvious. The most likely cause of this visual disorder seems to be a disorder of accommodation and fixation. There can be no doubt but that this indistinctness of vision plays a part in the production of the visual hallucinations, or more correctly, illusions. The most important part, however, we must admit to be the mental alteration, namely, the peculiar dipping down to lower levels of consciousness—if this term may be permitted—a condition of mental dissociation analogous to dreaming or to the hypnogogic state, in which hallucinations are also present. And we all know that in the state preceding sleep our vision becomes indistinct, as everyone has experienced when trying to read a book while having difficulty in keeping awake.

We will now return to the case of Miss G., and to the experiments on the process of apprehension. We wished to see whether a short exposure of letters or words or pictures was

<sup>1</sup> An interesting feature about the case were quite marked variations in the blood-pressure, distinctly perceived by the touch. But I was never sufficiently satisfied to declare that they were synchronous with these periods. On one occasion Dr Amadon established the fact that the fundus, which in the beginning of the ophthalmoscopic examination appeared normal, later was much paler.

sufficient for her to apprehend correctly. We used for that purpose a small screen of a photographic apparatus, the exposure of which varied somewhat between one-tenth and one-fourth of a second. Among seventy tests we found that sometimes we obtained, even with the shortest exposures, remarkably good results, which did in no way differ from the normal. This was especially the case with simple letters or with words. At other times the results were remarkably poor, and again the patient hallucinated. The influence of the clearness of the object was evidently of some importance. Thus, when an indistinct bird was shown, she said, "I see three cows in a field and a man coming along with a rake over his shoulder." Bonhoeffer, in studying his alcoholic deliria, has pointed out that by means of the *æsthesiometer* we sometimes get normal, again very bad results; in fact, his findings are perfectly analogous to ours. We may say that, from time to time, there is a most profound inability to apprehend, but that this is due entirely to the specific delirious alterations, the dipping down to lower levels of consciousness; while at other times we obtain normal results.

Somewhat more complicated is the study of the retentive faculty (*Merkfähigkeit*). When we gave the patient eight consecutive figures to repeat, she was able to give on an average about four; a few times, however, she gave seven and six, sometimes none or only one (nineteen tests). It is possible that seven and six represent her normal limit.

Other tests were the following. The patient was given pairs of words—(1) words connected by habitual association, such as "bread and butter"; (2) pairs of words connected by internal association, *e.g.* "head—hair"; (3) pairs of words which were not connected at all, such as "screen—ball." After times varying from thirty seconds to two minutes, thirty minutes, an hour, or even one or two days, she was given the first word and had to supply the second. We found that she was unable to retain words which were not connected, but we made few experiments with these. Among the words with internal connection she retained 31 per cent.; among those with habitual associations 57 per cent. It was generally found that when she was able to retain the words for thirty seconds she also could retain them for much longer periods, and the results with habitual associations were even strikingly good when she was asked two

or three days afterwards.<sup>1</sup> In this connection we may also mention some experiments with pictures. Three days after she had been shown certain pictures she was able to pick out correctly the five shown among twelve. And similar evidence of her ability to retain impressions was seen from day to day when questions about incidents of former interviews were asked. I doubt whether the results would have been the same in alcoholic delirium, for which Bonhoeffer claims such a memory defect, although he is not very explicit about it. At any rate, in view of these results, it seems very questionable whether we can speak in this case of a memory defect independent of the general clouding of consciousness. It might very well be that in alcoholic deliria, which have many points of relation with Korsakow's disease, there exists an independent memory defect, while this is not true in cases here under consideration. Finally, experiments similar to those recorded in relation with the Korsakow case were made, *i.e.* the patient had to tap every time a three occurred in a column of figures read to her. She omitted 34 per cent. These tests were made at two different periods—(1) when the delirious traits were more in the foreground; (2) when the retardation was more pronounced. During the former there were present 16·5 per cent. omissions, and 4·8 per cent. slow reactions; during the latter, 51·5 per cent. omissions, and 4·8 per cent. slow reactions.

If we summarise the clinical picture of these drug deliria, we find in the first place on the physical side invariably a coated tongue, a foul breath, sordes at times. We also find occasional slight febrile movements, sometimes unsteadiness of the gait, increase of reflexes, and some slight, but quite inconstant, tremor. The speech defect I am inclined to attribute in part to the bad condition of the mouth, in part to the clouding of consciousness, because it is very striking how much better these patients speak when they are aroused. There is no cyanosis and no flushing; on the contrary, the complexion of these patients appears rather pasty.

On the mental side we find first of all a certain dulness and hebetude, so that it is at times difficult to arouse these patients, while at the same interview it may be quite easy; and we have

<sup>1</sup> These word-pair experiments were made with twelve different word-pairs on five different occasions.

repeatedly noted that in spite of a marked dulness, unobtrusive noises may be commented upon. In harmony with this dulness is the fact that we often find a certain drowsiness even in the mildest cases. We shall later return to this.

The most marked alteration is a constant tendency to dip down to a lower level of consciousness. This seems to me a more correct formulation than to speak of an attention disorder, which term is used, for example, for the very different alteration underlying flight of ideas; although it is to be expected, and experience actually teaches us, that the lowering of consciousness which we here speak of should lead to an attention disorder, as a partial secondary manifestation, which then, of course, presents itself in a very different setting than that which produces a flight of ideas. When the consciousness sinks to this lower level we have a condition somewhat akin to sleep, inasmuch as there is a general dissociation; spontaneous trains of thought arise, not connected with the outside world or with reality, very similar to dreams. At the same time there are hallucinations of various senses, more especially sight, hearing, and touch. These hallucinations may be produced artificially by rubbing the eyes; they are also well observed if the patient is made to describe pictures or to read. We have seen that the visual hallucinations, or better the visual illusions, are in part at least due to an indistinctness of vision which we have reason to attribute to insufficient accommodation and fixation. However, the essential factor in the production of these hallucinations is evidently the general dissociation for which we find an analogy in the hypnagogic hallucinations and in dreams, and indeed it seems not improbable that hallucinations are most frequently produced by a dissociation of some kind or other.

It should again be emphasised that this, we might almost say, specific delirious tendency to dip down to a lower level of consciousness, is but a tendency, and that the patient can usually be roused, often to strikingly good, connected activity, as was shown in all our patients, especially well in the tests applied in the case of Miss G. The paraphasia seems entirely due to the lack of attention, the inability to concentrated activity as the result of the specific delirious alteration, as Bonhoeffer has shown.

The disorientation must also be explained on the ground of

this delirious change, and we have seen that in these drug cases a memory defect, independent of the specific alteration, can probably not be made responsible for this disorientation. But one thing should be mentioned in this connection. We have been struck with the fact that delirious experiences and delirious interpretations are held with remarkable tenacity, even during the convalescent stage, at a time when the patient is otherwise perfectly clear, and it is not improbable that this peculiar tendency, from an explanation of which we would refrain, is to a great extent responsible for the lack of correction which one would naturally expect in such patients who from time to time can be aroused to a connected mental activity.

As we have stated, the retentive faculty, or the memory for recent events as such, is probably not altered independently, and the same may be said in regard to the memory for old events.

The train of thought shows some characteristics which resemble those of flight of ideas, and are due, as we have said, to the incidental attention disorder, while at other times the connection is retained for considerable periods of time. What makes the utterances of the patient at times so incomprehensible to us is not this tendency to flighty turns, but rather the fact that delirious experiences are related with which we are not acquainted, and it is further made incomprehensible by the very frequent paraphasic elements.

The mood is often indifferent, but we have seen in one case a certain euphoria, again a certain whining depression, some indications of apprehensiveness, but never fear. So far as the motor side is concerned, we may find a certain restlessness or disinclination to move, but all this seems incidental to the essential delirious alteration: as a rule it shows nothing very pronounced.

We will finally compare with this picture that of the alcoholic delirium as Bonhoeffer describes it. According to this writer, this psychosis presents in 80 to 90 per cent. of the cases the following characteristics. The patient moves about a good deal, and is constantly occupied. His face is congested, his expression anxious. Often he shows marked fear. There is a very pronounced tremor, profuse perspiration. The gait may be somewhat uncertain, and there is ataxia of speech. We may add here that he mentions occasional eye muscle disorders,

which are, however, slight; and, retrospectively, the patients may speak of double vision.

The patients do not appear dull, and even at the height of the delirium they can be demonstrated to students, and the impression made on them is that the patient's manner of reaction is not markedly different from the normal; but the examiner finds that it takes some effort to hold the patient's attention. On a more careful examination, Bonhoeffer established the following. It is possible at any time to force the patient to a maximum degree of attention which does not differ from the normal. This may be shown, for example, by experiments with the æsthesiometer. A conversation with the patient also tends decidedly to raise his attention to a certain level, but when he is left to himself there is a constant tendency for the attention to reach a lower level, at which time the normal train of thought ceases, and the arising ideas show a marked tendency to become projected, as it were, as hallucinations. During an examination, when the attention is raised to a higher level, hallucinations are very few or totally absent, and the diminished attention shows itself chiefly by signs which are very similar to those of a normal inattentive state, such as a paraphasia similar to the fatigue paraphasia.

The memory for old events is not interfered with, and simple calculations are done well, as are all habitual tasks; but where a concentration is needed, and combinatory efforts are required, the patient fails. The retentive faculty, however, is markedly altered. On the ground of these deviations, Bonhoeffer explains the disorientation which in these cases is very marked. He also mentions in this connection a decided suggestibility and a marked tendency to confabulation, which we all know so well from our experience with Korsakow cases. From these confabulations he justly separates those which arise from hallucinations.

Bonhoeffer devotes considerable space to the hallucinations. He raises the question whether central or peripheral causes give rise to them. Meynert has claimed that in deliria the projection systems were at fault, and others had found various disorders, such as amblyopias (Magnan), retracted field of vision (Kruckenberg), disturbance of colour sensibility (Galezowsky). But Bonhoeffer points out how, on careful examination, he was

unable to find any of these changes, except perhaps in colour vision. He is of the opinion that peripheral changes, if they are of any consequence at all, have to be given a very subordinate place in the production of hallucinations. He mentions casually Mendel's claim that disorders of accommodation have something to do with visual hallucinations, but he takes no position in the matter. In describing the many mistakes which such patients make in reading, he says, however, that possibly the difficulty of convergence may partly cause this disorder, since he obtained better reading with monocular vision. He points out the well-known fact that the hallucinations in delirium tremens are apt to be combined, so that entire scenes are hallucinated; and he emphasises the frequency of the illusionary character of hallucinations, which are, after all, frequently a projection of the patient's thoughts. Just as Liepmann, so Bonhoeffer found artificial hallucinations produced by pressure on the eyeball, and hallucinations were also produced by looking at pictures, or by the reading tests.

Now the deviations from this picture are found either in complications with other psychoses or with epilepsy; but what interests us here especially is his description of the more severe cases. Such patients are more difficult to fix; finer tests cannot be applied. They are duller. The motor excitement is coarser, more elementary, the cyanosis is more marked, sweating and anxiety greater, the speech like that in meningitis. Eye muscle palsies are more frequent, as are various other paralytic phenomena. Such cases are very apt to terminate fatally.

If we now compare the two pictures, that of our deliria, and that of the alcoholic delirium as described by Bonhoeffer, we find, in the first place, that that which we have called the specific delirious alteration is present in both. The hallucinations are the same, and here, as well as there, it is easy to produce artificially these hallucinations; they are seen when pictures are described, and the results of the reading tests, *e.g.* are practically identical.

But all this we only find by a careful analysis, whereas superficially the two states differ so much that one would never be inclined to mistake the one for the other. That is due, in the first place, to the fact that we find in the alcoholic delirium the dilatation of peripheral vessels, and a tendency to cyanosis, and



often evidence of anxiety or fear. The pulse shows more marked alterations in alcoholic deliria. I am inclined to attribute these differences to the fact that the alcoholic delirium attacks persons who are chronic alcoholics, and whose cardio-vascular system, therefore, shows marked degenerative changes.

A further difference is to be found in the tremor, which is very marked in the alcoholic states, slight and inconsistent in the drug deliria.

Above all, however, the general responsiveness of the patient is different. We have seen that, according to Bonhoeffer, the alcoholics do not appear dull, and often make a strikingly natural impression on a casual observer so far as their manner of reaction to questions is concerned. In contra-distinction to this, we find our patients presenting a certain dulness and hebetude, and it is much more difficult to rouse them than it is to rouse alcoholic patients. It was a very natural supposition to think that possibly this greater dulness might be due to a disorder of apprehension which was added to the delirious alteration, and it was for that reason that the experiments on apprehension were made. They showed us that this is not the case. One might, perhaps, say that we happened to see graver states, conditions of unusually great intensity, and that the more marked conditions of alcoholic deliria, such as Bonhoeffer describes, are quite analogous, but are fatal only for the reason that the cardio-vascular apparatus is weak in the alcoholic conditions. That this explanation is not sufficient, is shown by the marked tendency to drowsiness even in our mildest case, Miss G. Therefore it cannot be merely a question of intensity, but this hebetude seems to be a special feature of these deliria. For some reason or other it seems that although a high level of consciousness can be reached in both kinds of cases, the tendency to sink to lower levels is greater in the drug than in the alcoholic deliria.

To a certain extent the fact that the alcoholic patient is constantly busy may depend upon this same difference. Whether there is, in the alcoholic states, also a certain elementary motor excitability, I am unable to say.

We have above mentioned the fact that Bonhoeffer assumes the existence of a memory defect for recent events in alcoholic deliria. Our experiments in the drug deliria, although they per-

haps do not allow a general conclusion, speak against such an assumption for our cases. And we have also stated that it would not be improbable that alcoholic conditions should present such a change though it be absent in our cases, because we know how often alcoholic deliria run into conditions of Korsakow's psychosis.

There is another symptom which Bonhoeffer mentions, the nature of which is as yet uncertain, viz. the great tendency to confabulation which he found in the alcoholic deliria. The "confabulation" which occurred in our cases appeared to be due entirely to the spontaneous trains of thought which were analogous to dreams, and which in part were externalised as hallucinations. We have, therefore, throughout our descriptions, spoken of the patients "relating delirious experiences." The fact that defects in the retentive faculty seem to have some relation to true confabulation, would suggest the possibility that the absence of confabulation and the absence of a defect of this nature were related; and, conversely, the lack of confabulation might be used as an additional support for the claim that the retentive faculty is not interfered with.

We see, then, that although superficially the alcoholic and the drug deliria are so different that the casual observer would never be reminded of the one by looking at the other, they have nevertheless both the same nucleus, *i.e.* the specific delirious alteration, which is only marked by certain special features characteristic of one or the other.

**THE HISTOLOGICAL APPEARANCES OF THE CORD  
AND MEDULLA IN A CASE OF ACUTE  
ASCENDING PARALYSIS.**

By CHARLES WORKMAN, M.D.,  
Pathologist to the Glasgow Royal Infirmary ;

and

WALTER K. HUNTER, M.D., D.Sc.,  
Assistant Physician to the Glasgow Royal Infirmary.

THE symptoms in the following case were apparently those of an acute ascending paralysis, and from the clinical point of view it might therefore be regarded as belonging to the group to which the name of Landry's paralysis has been given. But the lesion found on microscopic examination was of the nature of an acute myelitis, though of somewhat rare distribution; and it would seem desirable to classify such cases under the term "acute myelitis" rather than that of "Landry's paralysis," even though the paralysis was acute and ascending. Landry's paralysis, as at present understood, is not a specific disease with a definite and known pathological lesion, but rather a grouping of symptoms which may be produced by various, and sometimes widely different, morbid conditions. The term, therefore, should only be used to designate a grouping of symptoms of which the lesion has not been determined, and for which a more definite designation is not possible.

The case we have to describe is, unfortunately, not so complete, either in its clinical history or its pathological report, as it should be; still, the appearances in the cord and medulla are somewhat striking, and present a form of myelitis of much interest and some rarity. And from this point of view alone the case seems worthy of being put on record.

The patient, a boy aged 16 years, was admitted into Hartwood Asylum on July 4, 1904, in a state of stupor, being unable either to apprehend or answer questions. For some months past he had been suffering with headaches. He was irritable, apathetic to most of what was going on round about him, and slow in all his movements. At times he had delusions. But from the date of his admission he steadily improved, both bodily and mentally, and by the month of September he was able to go for long walks,

and seemed to be enjoying life as any ordinary individual. By this time he was considered perfectly well mentally.

On October 5, in the forenoon, he complained of having pains in the frontal region of the head. The temperature was 100° F., and the tongue coated with a thick white fur. A dose of castor oil was given, and by the afternoon the patient said he felt better. In the evening, however, he was not so well again, and complained of feeling out of sorts. By midnight the temperature was 103°, pulse 110, and respirations 30. There was complaint of headache and of pains in the knees, but physical examination was negative, except for the presence of a tender spot in the abdomen in the right iliac fossa. The temperature remained high all next day (October 6), and by the evening there was paralysis noted in both lower limbs. The loss of power was not absolute, as the left leg could be slightly raised and the toes of both feet could be flexed and extended. There was no anæsthesia and the reflexes were present. But later there was complaint of a feeling of discomfort in the throat, localised as being about one inch below the cricoid cartilage. Articulation was not affected, and while the breathing was somewhat laboured, there was no cyanosis. On examining the throat the left tonsil was seen to be slightly inflamed, but no membrane was visible. The next morning (October 7) the temperature was normal, but now all the accessory muscles of respiration were in action and cyanosis was very evident. Articulation remained unimpaired. Death occurred suddenly at 3 P.M.

The post-mortem examination was practically negative as regards naked eye appearances. There was no enlargement of the spleen and no meningitis. A small capillary hæmorrhage, about an eighth of an inch in diameter, was visible on the floor of the fourth ventricle on the left side near to the *striæ acusticæ*, but otherwise there was nothing of special note in the nervous system.

The cord, medulla, and pons were examined microscopically. They were fixed in formol and stained with thionin and with hæmatoxylin and eosin. (Sections were also stained in various ways to demonstrate if any micro-organisms were present, but with entirely negative result. Cultures were not taken.)

Throughout the whole length of the cord there was a marked infiltration of round cells into the grey matter. This was most

abundant in the lumbo-sacral and dorsal regions, and rather less in the cervical. The infiltration, moreover, was present throughout the whole breadth of the grey matter, but much more dense at the base of the posterior horn and at the base of the posterolateral aspect of the anterior horns. Indeed, the area of greatest intensity corresponded closely to the distribution of the anterior and posterior central arteries. The white matter of the cord was unaffected, except that the peripheral arteries running through it showed infiltration into their perivascular spaces, but not into the tissues beyond that. The vessels of the cord were greatly dilated, and there were some slight capillary hæmorrhages into the grey matter.

The vessels of the meninges were also congested, but the infiltration round these was slight in amount and was mostly confined to the ventral aspect of the cord, near to the anterior spinal artery, and into the median fissure.

In the lumbo-sacral region of the cord no normal ganglion cell could be seen in the anterior horns, and even the number of degenerate cells visible was small. The cells in Clarke's column were less affected. In the cervical region the infiltration was not so intense, and there was a proportion of the ganglion cells, mostly in the lateral groups, practically normal. Many of these seemed slightly swollen, but their Nissl granules were still intact and fairly well differentiated. The majority of the cells, however, were small, pale, and without any granulation; and many others had doubtless entirely disappeared. The process of cell degeneration seemed to be very rapid, for there were very few cells to be seen in a shape intermediate between the almost normal cell and the pale cell, with no granulation at all. Certainly no cells were seen with the dust-like granulation so typical of a more chronic chromatolysis. Some of the ganglion cells had apparently been invaded by the infiltrating cells, for they contained round cells in their interiors, but this was not a marked feature of the section.

Throughout the whole of the medulla and pons the vessels were greatly dilated as in the cord, and showed the perivascular spaces packed with round cells. Otherwise the infiltration into the grey matter was not nearly so marked as in the cord. In the lower parts of the medulla (level of pyramidal crossing) the infiltrating cells were almost entirely confined to what remained

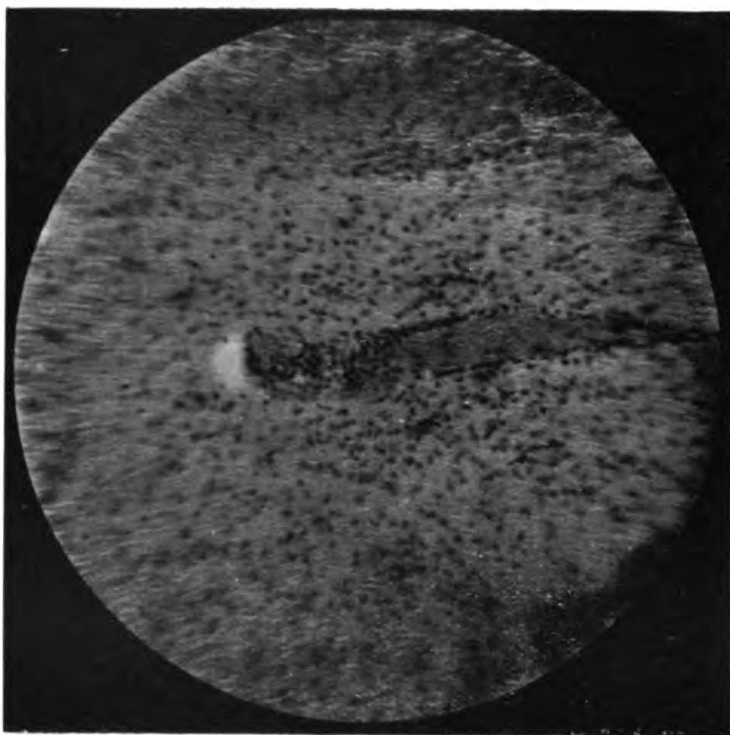


FIG. 1.

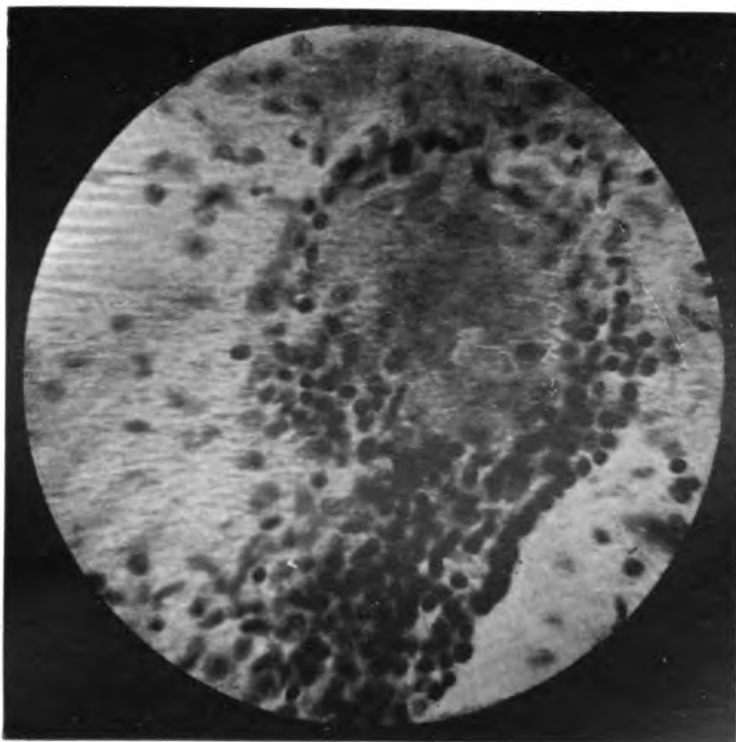


FIG. 2.



of the anterior horns : there was slight, if any, infiltration into the grey matter of the twelfth nucleus. Passing up the medulla, the ganglion cells of this twelfth nucleus were still seen to be quite normal, with no infiltrating cells round about them. But the infiltrated area could be traced upwards through the disappearing anterior horns to that part of the reticular formation round about the nucleus ambiguus. It seemed to be chiefly limited to this area, for the olive in front, the twelfth nucleus behind, and the lateral nucleus externally were all practically free from infiltrating cells. The ganglion cells of the nucleus ambiguus showed changes similar to those in the anterior horns of the cord.

The appearances in the pons were like to those in the medulla, but the amount of infiltration was rather less in the former. The infiltrating cells occupied a corresponding area in the reticular formation, and in the upper levels of the pons they were seen surrounding the fifth (motor) nucleus. Higher still (level of third nucleus) the area of infiltration seemed to have passed inwards, for it was most marked on each side of the middle line. The rest of the brain was unfortunately not examined.

The appearances were, therefore, those of an acute diffuse interstitial myelitis, limited to the grey matter of the cord, with the inflammatory process passing upwards into the grey matter of the medulla and pons. It was, indeed, a central myelitis, not, however, limited to an area round the central canal, for this part of the cord was comparatively unaffected, but with the intensity of the infiltration rather towards the lateral parts of the grey matter.

The process seemed to commence at the lower end of the cord and to pass upwards, for the infiltration was considerably more intense in the lumbo-sacral than in the cervical areas, and still less in the medulla and pons than in either of those parts of the cord. In the lumbo-sacral region, too, the almost complete destruction of ganglion cells pointed to that level as being first affected.

We have already said that this destruction of ganglion cells in the inflamed area seemed to be very rapid, and this is so, for the illness did not last more than forty-eight hours, and yet at the end of that time not a normal ganglion cell was to be found



in the lumbo-sacral enlargement. The absence, too, of cells in various stages of degeneration also suggests an acute process. The degeneration of the ganglion cells was doubtless due to the presence of a toxic agent in the circulation rather than to pressure from the infiltrating cells. It is well known how quickly ganglion cells may degenerate and disappear under the influence of certain toxins, and, on the other hand, how little they seem affected by the surrounding cells in certain other infiltrations. The exudation in this case, too, did not seem sufficiently dense to destroy the ganglion cells by actual pressure, neither was it specially centred round the ganglion cells.

The limitation of the infiltration to the grey matter of the cord is somewhat difficult to explain. But infiltrating cells most often follow the line of least resistance, and this is within the grey matter; possibly, also, the short duration of the illness prevented further extension. The area of greatest intensity was the same throughout the whole length of the cord and the same on both sides of the cord, though the exudation was invariably slightly greater on one side than on the other. And the area of the medulla and pons affected corresponded with the area of the cord affected, at least if we regard the nucleus ambiguus as the continuation upwards of the postero-lateral group of spinal cells. The fifth nucleus also continues upward the nucleus ambiguus and seventh nucleus cells.

As to the nature and origin of the infection—for the myelitis cannot be regarded otherwise than as an acute infective condition—we have little to go on. It is likewise difficult to determine whether it is of the nature of a septicæmia or a toxæmia. No micro-organisms could be found in the exudation after careful staining, and this favours the idea of its being a toxæmia; but, unfortunately, cultures were not taken from the throat, blood, or cord, either before or after death. The tonsillitis suggests the tonsils as the seat of entrance of the virus; and the possibility of the condition being a manifestation of diphtheria must also be thought of. Or it might even have been post-diphtheritic, for in the month of April the patient's brother had diphtheria bacilli discovered in his pharynx, and a week or two later the patient himself had a sore throat with some sort of exudation on the tonsils. But as he was on board a steamer at the time, and the steamer did not carry a doctor, no diagnosis can

be made regarding the nature of the sore throat. But in any case, as far as we know, the toxin of diphtheria produces no such interstitial exudation in the nervous system as was present in this case.

The nearest analogy one can find for this myelitis seems to be in the cellular infiltrations that are met with in rabies, a disease probably due to some toxin acting on the central nervous system.

And so we conclude that in this case also a toxin of some sort had gained entrance into the circulation, and that it acted on tissues unusually sensitive to such toxin (the history of the patient showed a marked neuropathic tendency), producing a degeneration in ganglion cells, and at the same time determining the infiltration of round cells, which was such a marked feature of the case.

In conclusion, we wish to express our indebtedness to Dr N. T. Kerr, Physician-Superintendent at Hartwood Asylum, who most kindly placed at our disposal the Asylum record of the patient's illness.

#### DESCRIPTION OF FIGURES.

FIG. 1.—Low power. Part of anterior horn of cord, showing dilated vessel with cellular exudation into the peri-vascular space and into surrounding grey matter.

FIG. 2.—High power. Shows a similar vessel with exudation into its peri-vascular sheath.

---

**TRYPANOSOMIASIS OR SLEEPING SICKNESS.**

By Major D. G. MARSHALL, I.M.S. (Retd.),

Lecturer on Tropical Diseases in the School of Medicine of  
the Royal Colleges, Edinburgh.

THE literature on this subject is now very extensive, and this article is written with the view of presenting, in a concise form, a review of our present state of knowledge of this most interesting disease.

*History.*—The earliest known description of the disease is an article by Winterbottom, published in 1803. In 1869, Guerin described minutely 148 cases occurring in Martinique during a period of twelve years, the disease having been introduced into the West Indies by imported African slaves.

The subject attracted little attention (though several cases occurring in natives had been recorded in this country) until 1900, when, owing to the opening up of trade routes, the disease, which had formerly been confined to West Africa, spread to Uganda, and caused enormous mortality among the natives, especially in the vicinity of the Victoria Nyanza.

In 1903 the epidemic had assumed such dimensions, and the high mortality caused so much interference with the development of the country, that a Royal Commission was appointed to investigate the disease, and from this time our more accurate knowledge of the etiology of the disease may be dated.

*Etiology.*—It is now generally admitted that the affection is due to the presence of Trypanosomes in the blood and cerebro-spinal fluid.

Trypanosomes were first described as occurring in the blood of rats by Lewis in 1877. Nepveu described what were apparently Trypanosomes in the blood of a man in 1895, but the proof of their connection with sleeping sickness dates from May 1901, when Forde, a colonial surgeon working in British Gambia, found worm-like bodies in the blood of a patient suffering from fever. In December 1901 he showed the slides to Dutton, who at once recognised the parasites as Trypanosomes. Later, Dutton found Trypanosomes in the blood of the European captain of a river steamer, who was suffering from

atypical fever. The man was sent home and the case carefully studied in the Liverpool School (1).

In September 1902, Manson, who had seen Dutton's case in Liverpool, was struck by the similarity of symptoms presented by an European lady who consulted him on account of fever contracted in Africa, and, as a result of repeated examinations, was enabled to demonstrate the presence of Trypanosomes in the peripheral blood. This patient died on Nov. 26, 1903 (2), death being preceded by marked signs of lethargy. (Fig. 1 is from a film of this patient's blood taken in Nov. 1902, for which I am indebted to Sir Patrick Manson.)

During the last three years about ten other European cases have been described, including one by Siend and Montier (3), and recently<sup>1</sup> three cases have been reported (4) as having occurred in Europeans prior to Manson's case, the cases terminating fatally in 1899, 1901, and 1903 respectively.

At this period (1901-2) numerous observers were independently investigating the disease in Africa, and the Trypanosome was not generally accepted as the prime factor in its causation. In particular the Portuguese Commission appointed to report on the disease in their own African territory came to the conclusion that it was due to a diplo-streptococcus which they found in 80 per cent. of their cases. They apparently overlooked the presence of Trypanosomes.

In Nov. 1902, Castellani, who was inclined to agree with the conclusions of the Portuguese Commission, found Trypanosomes in the cerebro-spinal fluid of a case, and later, by centrifuging the cerebro-spinal fluid, was able to demonstrate the presence of Trypanosomes in five out of fifteen cases.

The British Commission, under Bruce, on arrival in Africa in March 1903, were made acquainted with Castellani's results, and pursuing their investigations on the lines indicated by these results, were enabled to thoroughly work out the part played by Trypanosomes in the causation of the disease, and in their report (5) were able to tabulate certain definite statements, the majority of which are fully corroborated by Greig in the latest report of the Commission (6), a most valuable addition to our knowledge of the disease which will well repay perusal.

<sup>1</sup> *Annal de la Soc. Roy. des Sciences Méd. et Naturell*, Brux., f. xiv., 1905.

The statements, which are apparently fully proved by further investigation, are as follows :—

1. Trypanosomiasis and sleeping sickness are one and the same disease. The so-called Trypanosomiasis is simply the preliminary stage of fever.

2. Trypanosomes undoubtedly cause the disease.

3. Trypanosomes are carried by the *Glossina Palpalis* (Tsetse fly). No other insects, as *Stomoxys* or *Tabanus*, are concerned.

4. The distribution of sleeping sickness and *Glossina Palpalis* is identical.

5. The cerebro-spinal fluid of every case of sleeping sickness taken during life shows Trypanosomes.

6. Trypanosomes are not found in the cerebro-spinal fluid in any other disease.

7. The peripheral blood in all cases of sleeping sickness at some time or other shows the presence of Trypanosomes.

Among the very few statements in Bruce's report which, by the light of further investigations, are shown to be inaccurate, the most important is one regarding the function of the Tsetse fly in the development of Trypanosomes. He stated that in his opinion the fly acted simply as a carrier, and was not the seat of development of Trypanosomes. Recent researches (7) have, however, shown that very rapid development takes place in the stomach of the fly. See p. 40, and figs. 4 and 5.

In the latest report of the Royal Commission above quoted (6), Greig, as a result of extended observation and carefully conducted experiments on animals, makes the following categorical statements :—

1. The disease is a specific polyadenitis caused by the *Trypanosoma Gambiense*.

2. In addition to enlargement of the lymphatic glands, the blood shows a constant lymphocytosis at all stages of the disease.

3. Sleeping sickness is the last stage of the disease (Trypanosomiasis being simply the preliminary stage). It consists essentially in a polyadenitis plus signs and symptoms due to changes in the nervous system. The onset of these symptoms synchronises with the entrance of the Trypanosomes into the lymph spaces of the nervous system; this is accompanied by a rise of the mononuclear elements in the cerebro-spinal fluid.

4. Bacterial invasion, chiefly coccal, occurs in some cases, but only in the very last days of the sleeping sickness stage, and therefore cannot determine the onset of this stage of the malady.

Generally, these statements may, in our present state of knowledge, be accepted as correct ; for example, the opinion of the low pathogenic value of the diplo-streptococcus is confirmed by Lavarán as the result of a prolonged series of experiments, and the early affection of the glands with the easy detection of Trypanosomes in the fluid obtained from them by puncture in the early stage of the disease has also recently been corroborated by other observers.

The important practical question—Is Trypanosomiasis invariably followed by sleeping sickness?—can only be answered by the results of further observation and experiment.

The disease in natives is so chronic that only the close observation of a large number of cases from the first appearance of Trypanosomes in the blood to termination of the case by death from sleeping sickness or otherwise is necessary. The Royal Commission has had several natives under observation since 1903, but sufficient time has not yet elapsed for a definite conclusion to be reached. It is, however, certain that in all the European cases reported, the course of the disease tends to be more rapid than in natives, and Trypanosomiasis is invariably followed by death with symptoms of sleeping sickness.

#### *Description of Trypanosomes.*

It was formerly thought, and is still held by some, that *T. Gambiense* and *T. Ugandense*, *e.g.* the Trypanosomes causing the disease in Gambia and Uganda, were distinct species ; but pending further experiments, by which possibly they may be differentiated, it may be accepted that they are morphologically identical, and are one and the same species.

The Trypanosome (see Fig. 1) is 18 to 25  $\mu$  long, by 2 to 2.5  $\mu$  broad ; it consists of a body, flagellum, and undulatory membrane—the details of structure are well seen in specimens stained by the Romanowsky method. In the body about the centre is seen a large mass of chromatin staining red, the macronucleus ; near the posterior end is a smaller mass, the micronucleus or centrosome or blepharoplast. From this springs the

flagellum, about twice the length of the body, which, carried along the edge of the body with which it is connected by the undulatory membrane, projects from the anterior end.

When alive the parasite is seen to travel rapidly by a series of undulatory movements, flagellum first.

*Description of the Tsetse Fly (Glossina Palpalis).*

In the sub-family Glossina are included about twelve species, but so far only two are known to be concerned in the propagation of disease: *G. Morsitans* and *G. Palpalis*. The former in connection with Nagana (or cattle Trypanosomiasis); the latter acting not only as a carrier, but as a seat of multiplication of the Trypanosome of sleeping sickness.

*Habitat.*—Tsetse flies show a marked preference for certain areas, generally known as “fly belts,” which are characterised by the presence of water (rivers, or lakes), with abundance of shady shrubs in the close vicinity. They are practically never found except in the “fly belts”; and as the fly will not travel far from water, these belts are often of very limited extent.

The fly feeds only during the daytime (some species are said to feed both day and night); the natives know this, and endeavour to pass through the fly belt, if possible, during the night.

*Appearance of Glossina Palpalis.*

This, the darkest of all the Glossina, is about the size of an ordinary house fly. There are two chief characteristics which in common with the other Glossina render it easily distinguishable from other flies as the Stomoxys, which closely resemble it. These are the position of the wings when at rest and the wing venation.

The wings, which project slightly beyond the abdomen, are folded over one another like the closed blades of a pair of scissors. This position of the wings is not seen in any other flies except the Glossina.

*Wing Venation.*—The fourth vein, just before it meets the transverse vein, makes a distinct bend backwards. This is shown in Fig. 3, in which for comparison the wing of Stomoxys is also figured. This venation is absolutely confined to the Glossina.

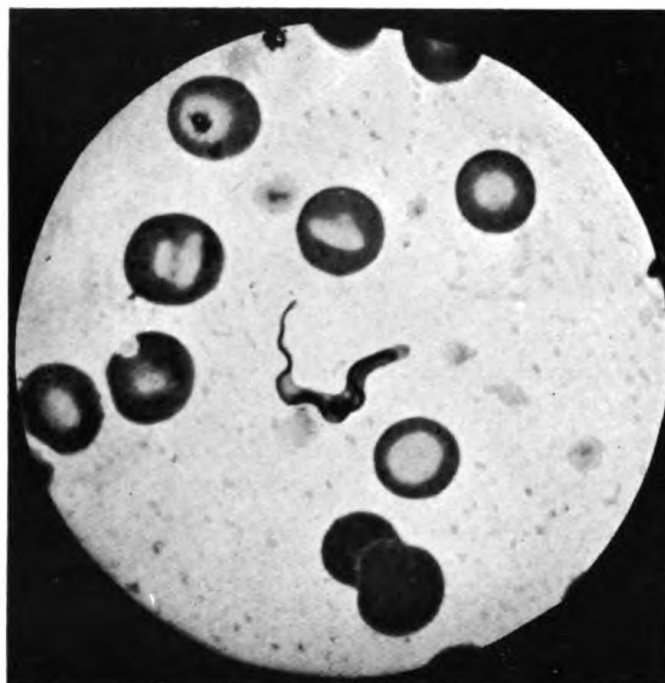


FIG. 1.

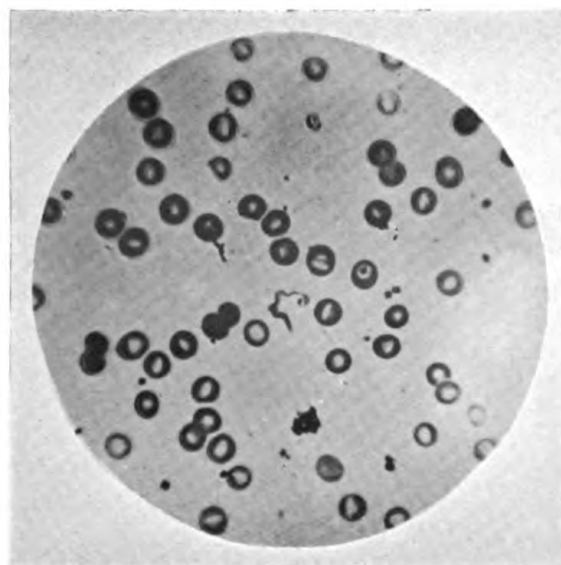


FIG. 2.





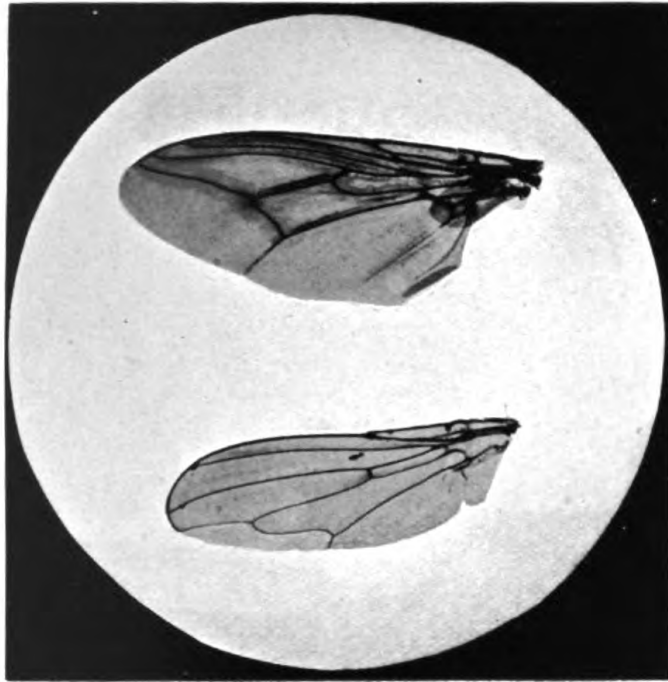


FIG. 3.



FIG. 4.



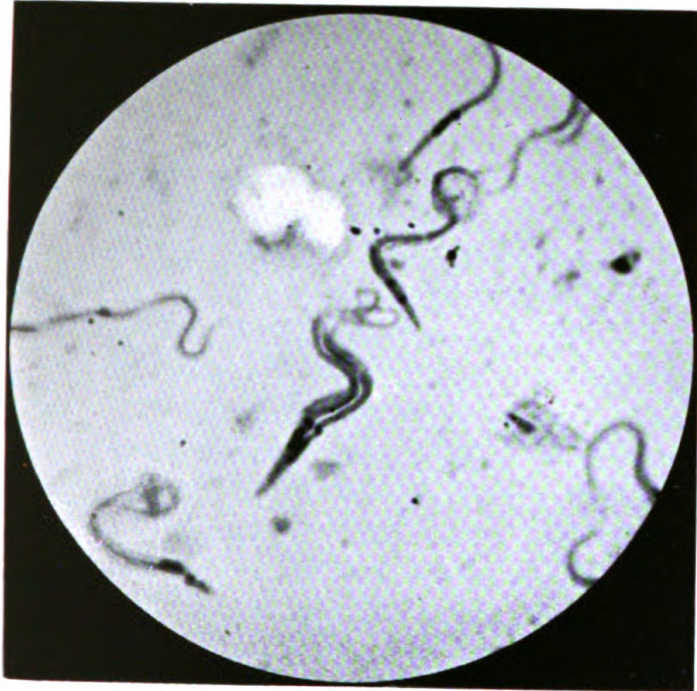


FIG. 5.

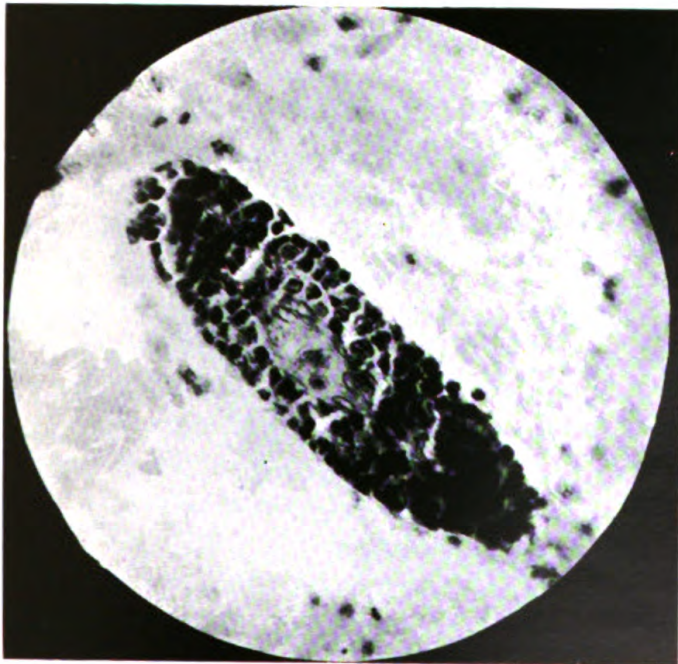
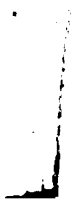
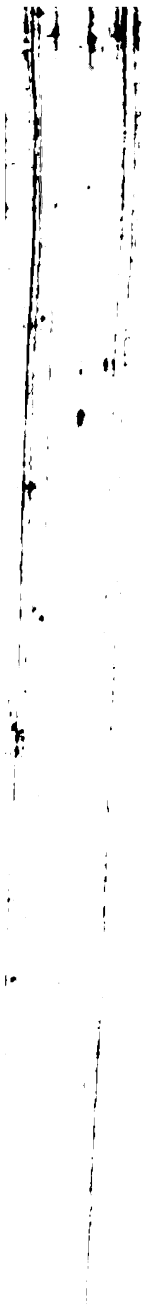


FIG. 6.



*Multiplication of Trypanosomes in the Stomach of the  
Glossina Palpalis.*

In the previously quoted (6) Report of the Sleeping Sickness Commission, Gray and Tulloch describe the experiments by which they showed that the Tsetse fly does not act simply as a carrier. Figs. 4 and 5 show the appearances in the stomach contents of a fly twenty-four hours after it had fed on an infected monkey. At the time of feeding, the monkey's blood only showed a few Trypanosomes—one in about six or eight fields—so that the enormous increase in number is very apparent. It is also interesting to note the varied appearances presented by the Trypanosomes. Some simply oval bodies with two nuclei, but no sign of a flagellum. Others elongated, long and narrow, also without definite flagellum. Others fully formed, and others again in the process of multiplication by longitudinal fission. These appearances closely resemble those found by Novy and MacNeal in Trypanosomes grown on artificial media.

Koch has recently published (7) a note in which he describes similar appearances in the stomach of the Tsetse, and suggests that some of the forms of Trypanosomes are male and others female, and that the development corresponds in some degree to that in the sexual form of the malarial parasite.

**PATHOLOGY.**

The changes occurring in the disease have been very fully described by Mott (8). They are slight and practically confined to the nervous system. The changes are identical in Europeans and natives.

*Naked Eye Appearances of Brain.*—There is often some flattening of the convolutions; the superficial vessels are injected; there is increase of the sub-arachnoid fluid, which is cloudy, giving a ground glass appearance to the membranes. These conditions are more marked at the base.

If the examination is made shortly after death, smears from the brain will often show Trypanosomes, but they are rapidly absorbed after death and are not likely to be found if the examination is delayed for more than six hours.

*Microscopically.*—The appearances are those of a chronic meningo-encephalo-myelitis, the chief feature being a filling of the

perivascular spaces with large and small mononuclear leucocytes. There is also some glia cell formation.

The mononuclear infiltration is well shown in Fig. 6, a section of brain from a very chronic case. In addition to the small and large mononuclears, there are larger cells—plasma cells of Marscholko—and large granular cells described by Mott as “Morula” cells.

This appearance, which is not confined to the brain, but is found in the nerve roots, nerves, and the viscera, resembles that found in many other chronic nervous affections, as general paralysis, disseminated sclerosis, etc.; but Mott states he has seldom found “Morula” cells in other forms of nervous disease except sleeping sickness.

Until recently, little attention has been paid to the other organs, but Greig has shown in his report that the stomach invariably presents a remarkable appearance, being studded with small ulcerating hæmorrhagic areas varying in size from a pin's head to a pea.

A coloured plate representing the appearance will be found opposite p. 266 of the report.

#### SYMPTOMS AND DIAGNOSIS.

During life the blood, lymphatic glands, and cerebro-spinal fluid show distinctive changes. These will be considered before describing the symptoms generally.

*The Blood.*—The changes here are :—

- I. Increase of mononuclear elements.
- II. The presence of Trypanosomes.

I. This is early manifested and persists throughout the course of the disease; there is no increase in the total number of leucocytes, the average count being 8000 per c.mm., but the polymorphonuclears are reduced to about 30 per cent., with a corresponding increase in the number of mononuclears.

Until late in the disease there is no marked diminution in the number of red corpuscles or of hæmoglobin.

In natives there is almost invariably an increase in the eosinophiles to 6 to 8 per cent.; but this has no connection with the disease, being due to the presence of intestinal parasites.

## II. *The Presence of Trypanosomes.*

The discovery of Trypanosomes in the blood is often a most difficult matter, and there is no doubt they are frequently missed—one writer has stated that finding them in an ordinary film is "always more or less a matter of chance."

The reason for this is that they are usually present in very small numbers, often not more than two or three in a slide, and therefore in stained slides may be easily overlooked, or only found after a protracted search. Much time is saved by using a low power in the search. The appearance, with which it is necessary to become acquainted, is shown in Fig. 2.

They are more easily found in fresh blood, as they attract attention by their active movements.

They are not constantly present in the peripheral blood. This is one of the most marked features of the disease. Examination of the blood may show their presence in increasing numbers up to a certain date, when they suddenly disappear, to reappear after a period of a few days, or, it may be, several months.

Working on Avian Trypanosomiasis, Novy and MacNeal have shown (9) that in suspected cases, in which examination with the microscope failed to show Trypanosomes, they were able to reveal their presence by cultural methods in 44 per cent.—in doubtful cases of human Trypanosomiasis, this procedure should be adopted, in addition to the inoculation of susceptible animals, as the ape or dog. Guinea-pigs are unsatisfactory.

### *The Lymphatic Glands.*

In a letter read before the Royal Society, 5th May 1904, Greig stated that, acting on a suggestion of Mott, he had examined the contents of the lymphatic glands in many cases of sleeping sickness and invariably found Trypanosomes.

Since then the subject has been more fully worked out, and it has been proved that examination of the glands in the early stage is a far more easy method of revealing the presence of Trypanosomes than examination of the blood. As, while they are, as previously stated, often present in very scanty numbers in the blood, they are almost invariably plentiful in the glands.

At first, the glands were excised, but this was found to be unnecessary. It is quite sufficient to puncture the gland with



an ordinary hypodermic needle, suck up a little fluid, and blow it out on a slide.

By this procedure, Greig found that often 50-70 per cent. of the natives in certain districts of Uganda harboured Trypanosomes ; while quite recently another observer, working on the Congo, has by the same means found 80 per cent. of the natives to be affected.

*The Cerebro-Spinal Fluid.*

In the later stages of the disease, examination will usually reveal the presence of Trypanosomes in the cerebro-spinal fluid, but they are usually in small numbers, and it is necessary to draw off about 5 c.c. of fluid and centrifuge it.

For a week or two before death, careful examination of the fluid often fails to show them, and it is during this time that bacteria are found in the fluid. This is an interesting fact, as Novy and MacNeal have demonstrated by their cultural experiments that Trypanosomes will not grow in the presence of bacteria.

The cerebro-spinal fluid also shows, from the earliest stages of the disease, the presence of lymphocytes—the numbers rapidly increasing towards the end. In the early stage there may be about 20 per c.mm., rising to 700-800 in the last stage.

The symptoms throughout are of a most irregular character.

The incubation stage may be very short or prolonged. Cases have been recorded in which natives did not manifest any signs of the disease for eight years after being exposed to infection ; in Europeans the onset is usually more rapid.

The associated fever is in the early stages also of a most irregular type ; it may be intermittent or remittent, persistent or showing periods of high fever, lasting for several weeks, alternating with irregular periods of apyrexia.

Death may ensue within a few months, especially during an epidemic, or may be delayed for several years.

The course of the disease may, for practical purposes, be divided into two chief stages.

*A.* An early stage (Trypanosomiasis), in which the Trypanosomes are present in the blood and lymph glands.

*B.* A later stage (sleeping sickness), in which the Trypanosomes invade the lymph spaces of the nervous system.

*Early Stage.*—As before stated, the symptoms in this stage differ very much in natives and Europeans. In the former, Trypanosomes may be present in the blood for several years without producing marked symptoms, while in most of the European cases recorded the fever due to the presence of the Trypanosomes has often been manifested within a few weeks or months.

In natives, one of the earliest signs is enlargement of glands, especially the posterior cervical. This has been long known among natives. In the old days, slave traders invariably rejected men from sleeping sickness areas if they presented enlarged glands.

The other marked features in this stage are :—

The irregular fever, which does not yield to quinine.

Vague pains in the chest.

Intermittent headache, lassitude.

Hurried respiration and quick pulse.

In most Europeans, a curious erythematous eruption tending to run into circular patches has been noticed.

Localised œdema.

In this stage the diagnosis depends on the finding of Trypanosomes in the blood and glands, and the resistance of the fever to quinine.

*Later Stage.*—Sleeping sickness. The earliest symptoms in this stage are very vague. They are chiefly a peculiar arhythmical tremor of the tongue, the hands, and upper extremity. The patient often complains of vague pains in the limbs and neck. The pulse is quick, 90-120, the respiration hurried.

The superficial reflexes are unaffected throughout ; the deep reflexes, at first exaggerated, are afterwards lost.

The patient gradually passes into a state of lethargy—at first there is simply sluggish action of the brain, a disinclination for exertion of any kind ; as the condition advances the patient assumes a somnolent condition. From this he is easily roused, but quickly relapses if left alone. In the last stage there is generally rigidity of the muscles of the neck with flexure of the lower limbs, extreme emaciation, which is sometimes masked by œdema, the skin is rough and dry, power over the sphincters is

lost. Saliva trickles from the corner of the mouth. Death results from exhaustion. The temperature for some days before death being usually subnormal.

The mind is usually perfectly clear until near the end, the patient being conscious of his condition, but occasionally, and this appears to be more common in Europeans than natives, there is delirium with maniacal excitement, or epileptiform attacks.

*Prognosis.* — Time and further investigation alone will show whether all cases of Trypanosomiasis proceed to the stage of sleeping sickness, or whether in certain cases, especially in natives of the endemic areas, a certain immunity is acquired. So far all European (except two or three still under observation) cases of Trypanosomiasis have ended fatally with symptoms of lethargy, and it may be held that, once the nervous system is invaded, death is inevitable.

*Treatment.*—Various drugs, as arsenic, chrysoidin, trypanoth, methylene blue, have been used with little result. At present in Africa the intra-muscular injection of arsenic is being extensively tried.

Better results are promised from the employment of serum obtained from immune animals. There is scope for further work in this direction.

#### DESCRIPTION OF FIGURES.

FIG. 1.—Trypanosome in blood film. European case.  $\times 1000$ .

FIG. 2.—The same.  $\times 250$ .

FIG. 3.—Shows above, wing of *Glossina Palpalis*, with the characteristic backward bend in the fourth vein. Below, for comparison, wing of *Stomoxys*.

FIG. 4.—Stomach contents of *Glossina Palpalis* twenty-four hours after feeding on a monkey infected with the Trypanosome of sleeping sickness. Note enormous number and developmental forms.  $\times 500$ .

FIG. 5.—The same,  $\times 1000$ , showing irregular forms and one large Trypanosome in process of longitudinal division.

FIG. 6.—Shows in centre cerebral blood-vessel surrounded by the mononuclear infiltration.

The whole of the illustrations have been specially made for this article. For the slides from which 4, 5, and 6 were photographed, I am indebted to Capt. E. D. W. Greig, I.M.S.; 5 and 6 are from photographs by Mr Richard Muir.

## LITERATURE.

1. "Note on a Trypanosome in the blood of man." Dutton, *Brit. Med. Journ.*, September 20, 1902.
2. *Brit. Med. Journ.*, May 30 and December 5, 1903. *Brit. Med. Journ.* April 30, 1904. "The examination of the tissues of the case of sleeping sickness in a European." Low and Mott.
3. "Recherche Bact. et Histol. dans un cas de maladie du sommeil chez un blanc." Sicard and Montier, *La Presse Méd.*, December 13, 1905.
4. *Annal. de la Soc. Roy. des Sciences Méd. et Nat.*, Brux., f. xiv., 1905.
5. "Further report on sleeping sickness in Uganda," by Bruce, Nabarro, and Greig. Published by Royal Society, November 20, 1903.
6. *Reports of the Sleeping Sickness Commission of the Royal Society*, No. vi., August 1905. Printed for H.M.'s Stationery Office by Harrison & Sons, London.
7. *Deutsch. med. Woch.*, November 23, 1905.
8. *Path. Soc. Transactions*, Vol. i., 1900. *Brit. Med. Journ.*, December 10, 1904.
9. *Journ. Infect. Dis.*, Vol. ii., No. 2, March 1, 1905.

---

## Abstracts

## ANATOMY.

**THE MODIFICATIONS OF NERVE CELLS, STUDIED BY NISSL'S**

(42) **METHOD.** (Les modifications des cellules nerveuses, étudiées par la méthode de Nissl.) CH. MOURRE, *Arch. gén. de Méd.*, Dec. 12, 1905.

THIS is a long paper dealing with the normal histology of the chromophile elements, and the changes in them, in various pathological conditions, chemical poisonings, toxæmias, etc.

The author concludes that the Nissl granules are unequally distributed through the cell, and do not constitute an essential part of it; they are subject, normally, to considerable variations, which are exaggerated in certain physiological conditions, and attain their maximum in pathological states. They are easily altered, but although showing many varieties of lesion, none of these are specific.

DAVID ORR.

**CHROMATIC PSEUDO-CORPUSCLES OF THE AXIS-CYLINDER.**

(43) (Pseudo - corpuscoli cromatici del cilindrasso!) PIETRO GUIZZETTI, *Riv. di Patol. nerv. e ment.*, F. 10, 1905, p. 473.

THE author refers to his observation, three years ago, of these bodies in a dog's spinal cord, fixed in sublimate-picric solution and

stained by Unna's polychrome blue and Grüber's orange-tannin mixture.

The axis-cylinders appeared for some distance completely coloured by the polychrome blue; other parts were yellow or reddish, and showed blue or blackish corpuscles. These were sometimes round, but more usually oval or fusiform, and lay along the long axis of the fibrils.

After using many fixatives and stains, and employing the tissues of various animals, the author is inclined to the opinion that these corpuscles are artefacts, principally on the ground of the almost absolute impossibility of demonstrating them by other methods than that detailed in the paper.

DAVID ORR.

#### ON CELL TYPES IN THE SENSORY GANGLIA IN MAN AND

(44) **THE MAMMALS.** (*Tipos Celulares de los Ganglios Sensitivos del Hombre y Mamíferos.*) S. R. CAJAL, *Trab. del Laborat. de investig. Biologicas*, Vol. iv., 1905.

CAJAL, in an interesting communication, demonstrates the superiority of his silver method over all others, and indicates that the formula used was his *second*, i.e. the impregnation with silver nitrate, after previous induration for 24 hours in alcohol abs. pure, or with the addition of two or three drops of ammonia. In his preparation, the spinal ganglia of man and the mammals are found to consist of the following types:—

I. The ordinary monopolar corpuscle, the expansion of which is arranged in a glomerulus.

II. Multipolar corpuscles having, besides the ordinary axon, short, thick dendrites with clubbed ends, which terminate within the capsule.

III. Multipolar capsules provided with fine expansions, ending in swellings or spheres of large size.

IV. Fenestrated corpuscles.

Besides these there appear in aged subjects:—

V. Corpuscles torn, ragged, or bristling with irregular appendices.

VI. Corpuscles strongly stained, showing no neurofibrils—probably dying or dead. It will be sufficient for our purpose to notice two of these varieties, those possessed of processes with clubbed ends and large spheres; and the fenestrated corpuscles:—

I. *Cells provided with processes terminating in capsulated balls.*—Cajal mentions that Huber published in 1896 a note announcing the discovery in a certain American frog of a corpuscle in the spinal ganglia having such terminations, but as this observation was not verified, it was supposed to be either accidental or a pathological condition.

Great was Cajal's surprise, on applying his silver method, to find in the ganglia of man a great number of cells which reminded him of Huber's observation.

Three varieties are noted :—

1. Having expansions with delicate filaments *which terminate within the capsule*.

2. Occurring chiefly in man or large mammals, as the horse and the ass, and having the terminations *outside the capsule*.

3. A mixed variety, having both intra- and extra-capsular expansions.

These cells are rare in the mammal, but common in man, especially in the plexiform ganglion of the vagus.

II. *Probable function of these cells with ball-like appendices*.—

What signification are we to attribute to these terminations in globes ?

Cajal finds this to be a problem of extreme difficulty, and he insists that this will not be solved until we discover special nerve arborisations, in contact with these spheres. He offers the suggestion that the sensory ganglia, in exception to the general rule that nerve centres are insensible, are provided with a sensory receptive apparatus, by means of which they can transmit to the cell, and so to the spinal medulla, any stimulus destined to regulate the sympathetic innervation of the blood-vessels.

*Fenestrated cells*.—It will be within the recollection of readers, that Cajal, in a recent *communiqué*, called attention to certain fenestrated cells which he found in the ganglia of the 9th nerve of dogs suffering from rabies.

This he considered to be a pathological condition exclusively found in rabid dogs. But as the result of a series of experiments which is still incomplete, he holds the view that these cells are normal in many mammals, and also, but more rarely, in man. After describing the various forms these fenestrations take, how they form loops, apparently sometimes multiplying the roots of the axon, sometimes appearing at the other pole of the cell, he suggests that they are homologous with the glomeruli of the ordinary cells, and that they therefore represent an organ whose object is to multiply the points of contact with the afferent nerve fibres. The exploration, he says, which is not yet complete, of the fenestrated system of the mammals, proves the existence in the spinal and cranial ganglia of a cell-type whose peripheric protoplasm extends itself in cords and network, apparently to meet the pericellular arborisation of the different nerves.

These cells are peculiarly rich in sub-capsular or *satellite* cells, concerning which Cajal hazards some very interesting opinions. He combats the idea held by Metchnikoff and others, that these satellites are simply neuronophagi, emigrated phagocytes, whose

sole function is destruction. He inclines to hold that they rather fulfil an important function in the modelling of the neuron; that they serve a similar purpose to that of the osteoclast in the building up of bone, with this difference, that in place of acting by erosion, their *modus operandi* is to stimulate the nutrition and growth of the neurofibrils, and in this way to determine important morphological changes in the shape of the body and processes of the ganglionic cells. It is suggested, in order to account for the diverse shapes produced during growth and in old age, that there is in the neuronal protoplasm a species of *anti-mitogenic* substance; that in maturity and in health this substance checks the mitosis of the satellites, but that in old age or ill-health the anti-mitogenic activity diminishes, and the dam, so to speak, which controlled the satellites is broken; they burst forth, increase and prosper; they penetrate the protoplasm and excite the neurofibrils which project outwards to the capsule, and so the peculiar appearance of the decrepit cells is produced.

All this is pure conjecture, and, as Cajal says, his experiments are not finished.

A. S. CUMMING.

#### ON THE DEVELOPMENT OF THE HIND-BRAIN OF THE PIG.

(45) O. CHARNOCK BRADLEY, *Journ. Anat. and Phys.*, Vol. xl., 1906, pp. 133-151.

THIS forms the second part of a communication, the first instalment of which has already been noticed in this Review (December 1905). Since 1886, when the late Professor His first called attention to the occurrence of a "Rantenlippe" in the hind-brain of the human embryo, several workers have expressed opinions, diverse in character, as to its formation and significance in different mammals. Some have suggested that a rhombic lip is probably present in all mammals; others have questioned its existence even in the human embryo. In pig embryos of nineteen and twenty-two days, there is no indication of the lip; but in an embryo 15 mm. in length, there is a very decided folding of the dorsal margin of the alar lamina. Sections across the hind-brain at this stage bear a marked resemblance to the figures given by His of a five weeks' human embryo, but with the difference that there is none of that flattening of the medulla at the widest part of the ventricle, which is so conspicuous in man. From the time of its first appearance, the rhombic lip is best developed in the region of the lateral recess of the ventricle, where it plays an important rôle in the formation of the tuberculum acusticum. It is difficult to imagine that, in the pig, the lip forms the olivary body, etc., in the manner described by His; it seems more probable that

the fasciculus solitarius becomes buried as a consequence of the migration of neuroblasts independent of the formation of the rhombic lip.

In a 23 mm. embryo there is a feeble development of the rhombic lip in connection with the cerebellum; but it is not so great as to lend support to the assumption that it plays more than a very small part in the formation of the cerebellum. The cerebellum is undoubtedly developed from a pair of lateral Anlagen; and its development is such that the question is again raised as to whether the hind-brain should be considered as consisting of two brain segments or only one.

In the pig, there is no foramen of Majendie during embryonic life; and it is very doubtful if the opening exists in the adult. The opening in the lateral recess of the fourth ventricle, on the other hand, appears at a comparatively early period. In an 80 mm. embryo, the posterior part of the recess has very attenuated walls; and in an embryo 100 mm. in length, the epithelium of the posterior wall has disappeared. In a 150 mm. embryo, the choroid plexus lies free in the subarachnoid space.

AUTHOR'S ABSTRACT.

## PHYSIOLOGY.

### ON THE GRADATION OF ACTIVITY IN A SKELETAL MUSCLE-

(46) FIBRE. KEITH LUCAS, *Journ. of Physiol.*, Nov. 9, 1905, p. 125.

THE extent of contraction of a many-fibred skeletal muscle might be varied by a similarly graded contraction of each individual fibre, or by the contraction of a varying number of fibres. Most of the evidence hitherto obtained favours the probability of the latter explanation. Keith Lucas experimented on the "cutaneus dorsi" muscle of the frog, in which the fibres run parallel, and can be easily counted and separated into groups. Groups of muscle fibres were stimulated directly with break induction shocks varying gradually in strength from minimal to maximal, and the contractions were recorded by means of a fine lever bearing a galvanometer mirror and focussing a beam of light on a moving photographic plate. After each experiment the muscle used was fixed and its fibres counted. Ten series of observations are recorded. A uniform increase in strength of the stimulus does not give a uniform increase in the extent of contraction, but the latter increases in definite steps, and the steps in the grade from minimal to maximal contraction are always fewer in number than the fibres in the muscle. A movement of the secondary coil of



1 mm. is often sufficient to produce a step, and it requires a subsequent movement through several mm. to produce any further extent of contraction.

The fibres in a whole muscle must have a wide range of excitability to the direct stimulus, and the gradation of contraction in it depends almost entirely on differences in excitability between the several fibres. The skeletal muscle-fibre appears to behave to stimulation like a cardiac muscle-fibre.

PERCY T. HERRING.

**THE LAWS OF ERGOGRAPHY, A PHYSIOLOGICAL AND MATHE-**

(47) **MATICAL INVESTIGATION.** (*Les Lois de l'Ergographie, étude physiologique et mathématique.*) Mlle. J. IOTKYKO (of Brussels), *Ann. d'Electrobiol. et de Radiol.*, No. 3, 1905.

V. *Physiological Signification of the Constants or Parameters.* Equations containing constants are, of course, in everyday use in the physical sciences. In them, however, the constants never vary, once they are established; in biology, on the contrary, they are affected by innumerable circumstances. Hence arises the far greater complexity of the sciences of life.

In the fatigue curve the constants  $a$ ,  $b$ ,  $c$  of the equation

$$\eta = H - at^3 + bt^2 - ct$$

represent the losses or gains in power at the end of a unit of time. Of these,  $b$  being positive tends to raise the curve, the other two,  $a$  and  $c$ , being negative, to lower it. The most probable interpretation of these parameters, and one which the experiments subsequently described tend to confirm, is as follows. The positive constant  $b$  is attributed to the action of the nervous centres, the action of which increases in ergographic work to cope with the paralysis creeping over the muscles. The negative parameters,  $a$  and  $c$ , are attributed to processes taking place in the muscle itself which produce a progressive diminution of the work. This interpretation is not arbitrary, being based upon our knowledge of physiological processes.

What does actually take place in a muscle which works to the point of fatigue? It is generally agreed that a muscle when fatigued consumes material different from that which it uses when fresh. When fresh, it draws upon its carbohydrates, the result being total combustion; any toxins which are produced are small in quantity, and are immediately consumed by means of the oxygen of the blood, destroyed in the liver and other glands, or eliminated by the kidneys. On the other hand, when exercise is prolonged to the point of fatigue, or there is an insufficient supply of carbo-

hydrates, then the albuminoids are consumed, many of the waste products of which are endowed with a high degree of toxicity. It is the accumulation of these substances in the organism which has a paralysing effect upon the muscles. It must not be thought that the carbohydrates are entirely exhausted before the consumption of the albuminoids begins; it is more probable that the two processes overlap, and that the accumulation of the toxic products always prevents our using up the muscle's whole stock of reserves.

This being the state of our knowledge before mathematics was called to our help, the author proceeds with her interpretation of the parameters.

The two negative constants,  $a$  and  $c$ , must necessarily correspond to the two chemical processes taking place in the muscle.

The latter must represent the loss of carbohydrates, seeing that in all motors the consumption of the combustible is proportionate to the time. The former,  $a$ , will then correspond to the effect of local intoxication by the toxins. It is very small at the beginning, but increases very rapidly (being multiplied by the cube of the time); and these characteristics accord very well with what we suppose to be the course of action in the consumption of the albuminoids.

The interpretation of the positive constant  $b$  as representing the action of the nervous centres is based on experiment, Mosso having established by means of his ponometer the law that effort increases with fatigue.

The writer now proceeds to examine one or two facts already established in the light of this interpretation of the constants. There are cases in which the curve becomes a straight line: in terms of the equation this would mean that the constants  $a$  and  $b$  disappear, the tracing being affected by  $c$  (consumption of carbohydrates) alone. Now, does this correspond with the facts? As an example, take a curve given by Mosso ("La Fatigue," p. 63; Eng. Trans., p. 99), which is, if we exclude the two first contractions, practically a straight line; we find that this curve is produced by electric excitation—that is, the action of the nervous centres (represented by the constant  $b$ ) is excluded. Moreover, electric excitation being painful, the weight used is lighter than usual; hence we can very well conceive that the demand upon the albuminoids will not be sufficient to affect the curve, *i.e.*  $a$  will practically disappear.

As a second example, consider what happens in the case of the straight line curve given by a frog's muscle when detached from the body. Here electric excitation is also used, which accounts for the absence of  $b$ . But as strong electric stimuli may in this case be used, it is difficult to understand the absence of  $a$ . Two facts are advanced by Mlle. Joteyko as supplying the required explana-

tion. 1. The straight line given by Kronecker as the expression of fatigue in a frog is found only when the weight is supported between the contractions; when this is not the case, the curve approaches a hyperbola. It is quite clear that under the former conditions the fatigue must be less. 2. The muscles of the frog differ from those of man in that they can by drawing upon the free oxygen of the air recover from fatigue, even when they have been removed from the circulation of the blood. It may then very well happen that, thanks to this elementary respiration of the fibres, the potency of the poisons of fatigue is much diminished. Indeed, the resistance of the fibres of a frog's muscle to all causes of alteration—anaemia, poisons, death—is well known, and they may therefore easily offer more resistance than man's muscles to the poisons of fatigue.

The rest of this section is devoted to a few general remarks upon the equation, and to pointing out that the constants are probably complex in their nature, *i.e.* due to the action of several causes acting conjointly.

VI. *Alcohol.* The second half of Mlle. Joteyko's paper is to be devoted to the account of a series of investigations devised (1) to test her interpretation of the parameters, and (2) to analyse the effect of various agents on the muscles by aid of the parameters.

The first agent investigated is alcohol.

The method of experiment was in all cases the same. The subject made several ergograms (rhythm, two seconds) at intervals of one to two minutes. He then rested long enough to allow the muscles to become completely restored (45 to 60 minutes). Then after having taken the substance under investigation he again described the same series of ergograms.

The first substance investigated was alcohol. The experiments were mainly intended to test the correctness of the interpretation of the positive constant  $b$ , for it is known that alcohol in small doses has an exciting effect on the nervous centres.

The writer proceeds to give a summary account of the literature already existing on the ergography of alcohol. An account of her own experiments follows, the exact figures being given in eight cases, and reproductions of the tracings in two. The typical alcoholic curve is found to be longer than the normal curve, but the height of the individual contractions is decreased. The total work done is increased, while the quotient of fatigue (the average of the contractions) is diminished. Individual differences with respect to alcohol are, however, very great, so that results quite the reverse of the normal are not infrequently obtained.

The constants of five of the most characteristic pairs of curves were calculated, and from a comparison of the figures thus obtained some important conclusions are drawn.

The constant  $b$ , attributed to the action of the nervous centres, is found to have increased in four out of the five cases. In view of our knowledge of the exciting effect of alcohol on the nervous centres, the interpretation of the one case in which  $b$  diminishes becomes of supreme interest. Here is Mlle. Joteyko's explanation. The constants are obviously complex quantities; thus  $a$  may be due to the action of several different toxins, any of which may vary independently of the others; if two, for instance, were to vary inversely, the parameter as a whole would clearly not be affected. Now suppose  $b$  to be made up of two components  $b_1$  and  $b_2$ ; let  $b_1$  be the effect of the centres properly so called, and let  $b_2$  be the action of any toxins, muscular or otherwise, which affect them during the course of the work. A slight dose of alcohol increases  $b_1$ , but it is conceivable that it might at the same time so much diminish  $b_2$  that its effect upon  $b$  should be on the whole to diminish it. Now when we turn to the actual figures to see if we can find anything which seems to support this hypothesis, we see that while in the other alcoholic curves  $a$  decreases at most to a third of its former value, in this particular one in which  $b$  has decreased it falls to one-thirteenth. If we suppose then what is from other considerations very probable, that toxins which have a paralysing effect upon the muscle have an exciting effect upon the nervous centres, the explanation offered will be seen to verify itself.

As has been said,  $a$  (action of toxins) diminishes in almost all the experiments (four out of five). It would appear from this that alcohol in small doses acts as an aliment, furnishing the muscle with easily assimilable materials which enable it to continue working without calling upon its own albuminoids. If we admit the peripheral origin of fatigue, this would also explain the fact that a little alcohol diminishes the *sensation* of fatigue.

*Effect of Alcohol upon the Rhythm.*—Maggiora has shown that an interval of ten seconds between the contractions allows the muscle to recover entirely from its fatigue, and admits of the contractions being maintained at their maximum. In the experiments devised with the view of testing the effect of alcohol on the rhythm, fatigue was considered to be held at bay if after forty minutes' work the curve showed no sign of declining towards the abscissa. When the work was done with a rhythm of eight seconds, fatigue appeared about the twenty-fifth minute; when the same experiment was repeated after the absorption of 30 grammes of alcohol, no trace of fatigue appeared. The effect of alcohol then is to quicken the rhythm at which work may be done without fatigue.

*Explanation.*—At first sight it might seem that the most probable explanation of this fact would lie in the exciting effect of alcohol on the nervous centres. But a closer investigation seems

to show that the action of alcohol as an aliment accounts more satisfactorily for all the phenomena. In the previous discussion it was shown that alcohol had generally the effect of diminishing the parameter  $a$  (attributed to the action of the toxins). Now if this is the case with ergograms written with a rhythm of two seconds, still more ought it to be the case with those written with a rhythm of four seconds, which approach complete indefatigability. If the constants are calculated for a normal curve and for an alcoholic curve with this rhythm, it is found that in the latter the constant  $a$  is reduced to one-thirteenth of its normal value, while the constant  $b$  is reduced to one-fifth. This result agrees with that of the experiment quoted above in the first series: the enormous diminution of muscular toxins coincides with the apparent absence of excitation of the nervous centres.

The most notable feature of the alcoholic curve is the enormous diminution of  $a$ . This appears explicable by the action of alcohol as an aliment, the muscle being enabled thus to refrain from making use of its albuminoids.

The writer concludes her study by pointing out that her results, established by the ergograph, agree well with those of Atwater, Duclaux, and A. Gautier, who, on the strength of their recent researches, have concluded that alcohol acts as a true aliment and even as a precious aliment, so long as one does not surpass a daily dose of 1 gramme per kilogramme of the weight of the body.

MARGARET DRUMMOND.

## PATHOLOGY.

### BAACTERIOLOGY OF ACUTE ANTERIOR POLIOMYELITIS. M.

(48) GEIRSVOLD, *Tidsskrift for den Norske Lægeforening*, Oct. 15, 1905.

IN the *Medicinsk Revue* for November 1901, Drs Carl Looft and H. G. Dethloft gave an account of previous bacteriological investigations in this disease.

Schultze, in a case, has found the meningococcus (*cf.* S. Auerbach, *Jahrbuch f. Kinderheilk*, Bd. 4, 1899). F. Harbitz and Bülow-Hausen found a diplococcus in the spinal fluid of one case examined post-mortem (*Norsk. Magazin for Lægevidenskaben*, 1898). F. Engel (*Prag. med. Woch.*, No. 12, 1900) found the staphylococcus albus in the spinal fluid of a boy who developed acute anterior poliomyelitis, but since the boy suffered from a chronic otorrhœa and an osteo-myelitis of the clavicle, he regards the find as accidental. Concetti (*Rev. Mensuelle des Maladies de l'Enfance*, 1900) has performed lumbar puncture in ten cases of the disease, in nine of which a bacteriological examination was made. In two

cases examined on the second and third days of the disease, the diplococcus of Talamon-Fränkcl was found; in one case examined on the seventh day the meningococcus of Weichselbaum.

Looft and Dethloft described two cases in the paper above referred to. A diplococcus was obtained from the cerebro-spinal fluid in both cases. The diplococcus, the characteristics of which are described in their paper, was like the meningococcus type of Heubner, or the coccus which Hunter and Nuthall (*cf. Lancet*, 1901, p. 1527) call meningococcus, type B.

In the present paper, Dr Geirsvold publishes an examination of twelve cases occurring during the epidemic which has been prevalent during the last few months in some districts of Norway. He has found a diplococcus almost identical with the coccus formerly found by Drs Carl Looft and Dr H. G. Dethloft.

*Characteristics.*—Bean-shaped diplococcus or tetracoccus, forming in bouillon chains 4-6 pairs. Division parallel to long axis of cocci. At first the growth is delicate, later in transferred cultures it is more luxuriant; then the colonies are more viscid.

On serum a delicate growth. Growth also on potato and in milk, which coagulates after some days.

The coccus is distinctly stained by the usual aniline stains and is Gram positive. It was not found in large numbers in the spinal fluid, which nearly always was clear and deposited a little sediment. The deposit contained mononuclear cells, but very seldom leucocytes.

For cultivating, one has to use great quantities (1-3 c.c. for each tube). If cultivating after the tenth or twelfth day after the beginning of the disease, he very seldom found any growth of the cocci; but in this case the spinal fluid microscopically examined showed micro-organisms in fairly large numbers.

The results of the experimental inoculations were:—White mice subcutaneously inoculated died after twenty-four hours; paralysis was sometimes noticed. Some other mice were ill during some days, then they rallied, but after twenty days a paralysis of the extremities was suddenly discovered. The paralysis was ascendant, and death occurred after extreme atrophy of the muscles had appeared. The same symptoms were noticed in rabbits. A sudden paresis of the posterior limbs after a long incubation.

The same coccus has also been cultivated from the throats of patients suffering from acute anterior poliomyelitis, and from persons living in places where the disease has not yet appeared. In all these cases the coccus was pathogenic for mice.

CARL LOOFT.

**CONTRIBUTION TO THE STUDY OF MENINGEAL TUMOURS.**

(49) (*Contribution à l'étude des tumeurs méningées.*) G. ROUSSY,  
*Arch. gén. de Méd.*, Dec. 19, 1905, p. 3211.

AFTER giving a description of the morbid histology of three meningeal tumours, the author discusses the nomenclature of such growths. He concludes that "tumours developed from the meninges, commonly called psammomata, angiolithic sarcomata, or meningeal endotheliomata, would be better described as sarcomata of endothelial type with hyaline or calcareous masses of vascular origin." He is of opinion that the concentric bodies so often found are due to the formation of buds by the proliferation of the inner coat of the vessels, with a subsequent hyaline or calcareous degeneration.

STANLEY BARNES.

**THE MECHANISM OF NERVE REGENERATION. (*Mecanismo de***

(50) *la Regeneración de los Nervios.*) S. R. CAJAL, *Trab. del Lab. de Investig. Biológ.*, Vol. iv., 1905.

PRELIMINARY notes of these results appeared in the June and September numbers of the *Boletín del Instituto de Bacteriología*.

It is well known, says Cajal, that when a peripheral nerve is severed, and the ends approximated, there appear in the cicatrix bundles of newly-formed nerve fibres, by means of which eventually communication is restored. How is the regeneration of the distal segment effected?

Cajal recounts at length the history of this great controversy. After detailing a very full and interesting series of experiments, which are profusely illustrated, he proceeds to give what he calls a rational interpretation of the facts.

The act, he says, of severing a nerve-cord, leaving a group of axons and cells of Schwann absolutely isolated from all central trophic influence, immediately awakens two defensive mechanisms. The first, for which the cells of Schwann are responsible, has for its object the destruction and reabsorption of the useless axon and myeline, leaving the way free for the new fibres; the second, which is the work of the axons of the central end, is for the creation and growth of the embryonal conductors, destined to re-establish communications. The cells of Schwann acquire great activity and proliferate enormously, and the greater part of the resulting cells, after completing the work of absorption, run their protoplasms together, forming a faintly outlined cylinder sprinkled with nuclei, the *cellular bands* of Búngner and Bethe. So that, as pointed out by Stroebe, the cells of Schwann represent real phagocytes which, like the leucocytes of the blood, can assimilate fatty particles and

can migrate. Cajal agrees with Stroebe in thinking that the greatest number of the granular interstitial cells of the peripheral segment are dislocated cells of Schwann. The *cellular bands* are found to be well formed within eight days.

What, then, is the function of these cellular bands. Cajal, who is a strong supporter of the theory of continuity, does not agree with Búngner, Bethe, and others, that they are destined to produce new axons on the distal side. He is inclined to think that they secrete what he calls *quimiotactical* substances, which excite amoeboid movements and a budding forth of the new axons, attracting secretions which draw the young fibres into the spaces which have been cleared for them. These secretions would be poured out on the seventh or eighth day, and would act as do the crystal tubes, full of attracting material, in Pfeffer's experiments on the spermatozooids of the cryptogams.

Thus it is seen that the cells of Schwann fulfil two functions: the first consisting in the destruction of the axon and myeline and its removal; the second in the production of *quimiotactic* secretions. Moreover, that this quimiotaxis does intervene has been almost demonstrated by the ingenious experiments of Forssmann; and to this attracting influence he gives the name of *Neurotropism*. A point in favour of this view is, as has been noticed by Vanlair, and is well seen in Cajal's illustrations, the behaviour of the young nerve-fibres on the proximal side of the wound. A large number wander from the path, to right and left; some turn backwards, and indeed they look as if they had lost their way. This happens before the seventh day, and is accounted for by the fact that the quimiotactic fluid is not at that time in action. As the fibres traverse the cicatrix, and approach the other side, their course is much more regular.

The reason why each nerve-fibre infallibly finds its proper place is, Cajal admits, not satisfactorily accounted for by either theory. He holds certain ideas on this question which he will develop later.

Cajal recurs in this paper to the subject of the satellite cells of the neurons. The neuron, he holds, is a complete entity only so far as the function of transmitting impulses goes; but for living, growth, the formation of branches, etc., it is dependent on the satellite cells. Between the neuron and its satellites there exists a sort of mutual association, comparable to the symbiosis of algæ and lichens.

*Formula Used.*—To 50 c.c. alcohol abs. add 2 or 3 drops of ammonia. Fix for 24 hours, then put the pieces in nitrate of silver, 1.50 per 100, in stove at 35° to 38°. Lastly, reduce by the pyro-formol mixture. As a rule, the pieces are kept under heat during five or six days.



Cajal considers that Marinesco, in his recent paper, has arrived at conclusions diametrically opposed to those which a calm study of the preparations should have led him to.

A. S. CUMMING.

### CLINICAL NEUROLOGY.

**JUVENILE TABES.** (Ein Beitrag zur Tabes in jungem Alter [Tabes (51) infantilis und juvenilis].) W. LASAREW, *Neurol. Centralbl.*, Nov. 1905, pp. 988 and 1047.

THE author points out that probably in some of the cases recorded as juvenile tabes, the diagnosis has not been correct. He criticises many of the cases published, and states that he has only been able to find in literature the records of twenty-three undoubted cases.

Affections which are specially liable to be wrongly diagnosed as juvenile tabes are: Friedreich's disease, cerebro-spinal syphilis, and a form of combined postero-lateral sclerosis, which has been described by Oppenheim.

The author records a case of juvenile tabes which has come under his own observation. The patient was a female aged 19 years, and presented signs of hereditary syphilis (Hutchinson's teeth, etc.). The symptoms were of three years' duration. The knee-jerks were absent; the Argyll-Robertson pupils were present; there were shooting pains in the legs; Rhomberg's sign was present; and there was diminished sensation on the trunk of "root type." The muscular sense (sense of position) was lost in the legs.

A review of the symptoms in juvenile tabes is added. The disease begins most frequently between the ages of ten and twenty: males and females are affected with almost the same frequency. In one-third of the cases, bladder symptoms (difficulty in passing water, and especially involuntary micturition) were the first indications of the disease. Headache (often simulating migraine) is another symptom not infrequently met with in early juvenile tabes; and in a few cases failure of vision (due to optic atrophy) has occurred as an early sign.

Many symptoms, common in tabes of the adult, occur in the juvenile form, viz. loss of the knee-jerks, Rhomberg's symptom, ataxia, shooting pains, sensory symptoms (analgesia, diminution of sensation), optic atrophy, changes in the pupillary reflexes, etc.

In conclusion, the author discusses the etiology of juvenile tabes, and points out the frequency of evidence of hereditary or acquired syphilis. In the twenty-four cases of juvenile tabes on which he bases his analysis of symptoms, there was undoubted evidence that the parents were syphilitic in eleven (the father

was syphilitic in seven cases, the mother in one, both parents in three). Many of the juvenile patients presented signs of hereditary syphilis. In two of the cases syphilis was acquired at a very early age—in one case at the age of five years through the kiss of a prostitute; in the other case at the age of four months through the kiss of a nurse. In only four cases was there no evidence of syphilis, hereditary or acquired. Thus there was evidence of syphilis in 84 per cent. of the cases. In the adult, tabes is much more frequent in males than in females; in juvenile tabes females are affected as frequently as males, since the majority of cases of juvenile tabes are due, at least indirectly, to hereditary syphilis.

R. T. WILLIAMSON.

**CHRONIC ANTERIOR POLIOMYELITIS, WITH THE REPORT OF**  
(52) **A CASE WITH NEUROPSY.** MOLEEN, *Am. Journ. Med. Sci.*,  
Dec. 1905, p. 1025.

IN the case recorded, the condition began suddenly with lameness in the left foot, followed two weeks later by wasting in the peroneal region, diminution of Faradic excitability and decrease of reflexes, the upper limbs, however, remaining unimpaired. Fifteen months later the face, tongue, and upper limbs (especially the thumb muscles) showed wasting and fibrillary tremors, and the reaction of degeneration was present in both upper and lower limbs. A careful examination of the spinal cord was made after death, and showed congestion of the small blood-vessels of the anterior horn, with degeneration of the anterior nerve roots in the lumbar region and numerous small hæmorrhages in the thoracic and lumbar regions (shown in a plate).

The question regarding the classification of cases of spinal muscular wasting is very fully discussed, and there are references to some twenty-three other cases of a similar nature that came to necropsy.

JOHN D. COMRIE.

**SPINAL HÆMORRHAGE; SOME OF ITS GENERAL PHASES.**  
(53) WILLIAM BROWNING, *Medical News*, Oct. 7, 1905.

THESE cases may be classed from an etiological standpoint in three groups: (1) traumatic; (2) secondary, originating from tumours, etc.; (3) spontaneous. Anatomically three divisions may be recognised: (1) epidural; (2) submeningeal; (3) myelic. The author regards meningeal or even intrameningeal as unsatisfactory terms.

The epidural venous plexus is the source of hæmorrhage in this locality. Ten cases are cited from the literature, in only three of which was there a distinct complaint of pain at the onset or sub-

sequently. In 20 cases of myelic hæmorrhage, in all but two pain was present. There seems much less tendency to impairment or loss of sensation in the epidural than in the myelic form. Girdle sensation was present in one of the epidural and in five of the myelic cases. Dissociated sensory loss was not observed in the epidural cases; the presence of this symptom "can be taken as *prima facie* evidence that it belongs to the myelic form." The distinction between the epidural and myelic types is of importance, for in the former, since the blood tends to collect posterior to the cord, without spreading up and down to a great extent, there is a reasonable hope of successful surgical intervention.

The subdural type of submeningeal hæmorrhage is unimportant except as a complication, likewise the subarachnoid. The subpial form appears at times independently, these cases closely resembling hæmorrhage into the cord. Myelic hæmorrhage (hæmorrhage into the cord) is capable of subdivision into several varieties. Of these, the central or tubular type is considered as almost synonymous with traumatic, since in traumatic cases the hæmorrhage so frequently takes on the elongated form.

The single or focal form of myelic hæmorrhage has been supposed to correspond to the ordinary form of cerebral hæmorrhage. It is, however, surprising to note that many of the cord cases occur before the usual period of life for cerebral hæmorrhage. Of 20 tabulated cases treated, only 7 occurred in persons over 45 years of age. Spinal and cerebral hæmorrhages are different processes. "Myelic hæmorrhage is not usually a result of the degenerations, but of the activities of life." By this the author does not wish to imply that myelic hæmorrhage is directly traumatic, although he points out the influence of flexion of the spine on the pressure of the cerebro-spinal fluid as observed on lumbar puncture, and concludes that this change in pressure must be transmitted to the vessels. An analytical table of the chief points in the 20 cases of myelic hæmorrhage is appended.

EDWIN BRAMWELL.

#### **A CASE OF SYRINGOMYELIA WITH DOUBLE OPTIC NEURITIS.**

(54) WEISENBURG and THORINGTON, *Am. Journ. Med. Sci.*, Dec. 1905, p. 1019.

THIS case is recorded fully because of the extreme rarity of optic neuritis in syringomyelia, the writers having succeeded in discovering only two other recorded cases of which they give the reference and an epitome. The present case occurred in a girl of 16, and the neuritis was so severe as to cause blindness in the right eye and ability only to count fingers at 10 inches with the left. The writers refer the optic neuritis to hydrocephalus.

JOHN D. COMRIE.

**DISSEMINATED SCLEROSIS; CEREBELLAR ATROPHY AND****(55) PSEUDO-SYSTEMIC SCLEROSIS OF THE SPINAL CORD.**

(Sclérose en plaques; atrophie cérébelleuse et sclérose pseudo-systématique de la moelle épinière.) G. CATOLA, *Nouv. Icon. de la Salpêtr.*, Sept.-Oct. 1905, p. 585.

THE patient was a man of 38 years, and the first noticeable symptom was trembling of the arms, which appeared five or six days after the onset of an attack of cholera. Later his legs became stiff, and eventually the usual symptoms of disseminated sclerosis made their appearance. There was scanning speech, horizontal nystagmus, and general intention tremors. Diplopia was present, but the reaction of the pupils was normal. The gait was ataxo-cerebellar and spastic, and the reflexes were exaggerated. Incontinence of urine and fæces supervened, and later delirium, passing into coma.

The histological examination showed very marked sclerosis of the pons and cerebellum. In the latter, Purkinje's cells were much diminished and there was a considerable increase of neuroglia. Two small patches of complete sclerosis were present in the *substantia nigra* of Sœmmering, but the remainder of the cerebral peduncles were normal. The median portion of the superior cerebellar peduncles was atrophied, but the middle and inferior peduncles were normal. Atrophy was also present in the olives and in the riband of Reil. In the cord there was a pseudo-systematic atrophy, chiefly situated in the antero-lateral columns. There was considerable thickening of the pia membrane, and the vessel walls were thickened throughout all parts of the cord, medulla, pons, and cerebellum. The author thinks that the relationship of these vascular alterations is causal, and that the cholera was an etiological factor. It is of interest to note that although the islets had such an anatomical restriction, the classical symptoms of disseminated sclerosis were nevertheless present.

A. F. TREDGOLD.

**FURTHER OBSERVATIONS ON THE RELATION OF LESIONS OF****(56) THE GASSERIAN AND POSTERIOR ROOT GANGLIA TO HERPES OCCURRING IN PNEUMONIA AND CEREBRO-SPINAL MENINGITIS. HOWARD, *Am. Journ. Med. Sci.*, Dec. 1905, p. 1012.**

THE writer records five cases of herpes, out of which three died, so that the condition of the ganglia concerned was fully investigated.

In case 1 (pneumonia complicating typhoid fever) there was

herpes on the right side of the nose, and, after death congestion, degeneration of the nerve-cells and infiltration of the connective tissue with leucocytes were found in the corresponding part of the right Gasserian ganglion, while the left ganglion was normal.

In case 4 (epidemic cerebro-spinal meningitis), with herpes of the right side of the face and neck and of the left upper lip, vestibule of the nose, side of the head, and soft palate, both Gasserian ganglia and the third right cervical ganglion were post-mortem found surrounded by exudate and having their nerve cells degenerated.

In case 5 (cerebro-spinal meningitis of actinomycotic origin), with trifacial neuralgia on the right side and herpes on the left side of the neck, the Gasserian ganglion after death showed no abnormality, though the right fifth nerve was markedly inflamed. Marked changes were found in the third and fourth cervical ganglia of the left side.

Commenting upon these cases, the writer shows the complete connection between herpetic lesions and ganglionic changes; in affections of the nerve roots, herpes apparently does not occur unless the change spreads to the ganglia.

JOHN D. COMBIE.

**CEREBRAL SYMPTOMS DUE TO ENCEPHALITIS AND THE  
(57) RELATION OF THIS DISEASE TO ACUTE ANTERIOR  
POLIOMYELITIS.** FRED. E. BATTEN, *Trans. Med. Soc. Lond.*,  
Vol. xxviii., p. 116.

THE writer deals with the clinical aspects of certain cerebral cases in children, which resemble those of infantile spinal paralysis in various ways. They set in suddenly, usually in summer, as acute anterior poliomyelitis does; they run a similar course and show a like tendency to more or less complete recovery. He regards them as almost certainly instances of polioencephalitis.

The cases are divided into three groups, according as the symptoms point to implication of (a) the cerebral hemispheres (P. superior), (b) the cerebellum (P. cerebelli), or (c) the nuclei or basal ganglia (P. inferior). *Polioencephalitis superior* may involve any cortical area. An instance is first given where the *frontal region* seemed affected. A normal child of two years and three months became suddenly ill with convulsions, vomiting, and fever; she lost her sight, could not sit up, and screamed for hours. After a week of unconsciousness she came round, but was found unable to walk or talk, and was dirty in her habits, very troublesome, and given to screaming. Two cases are then given of girls of three and five years respectively, in which the *Rolandic area* seemed

implicated. In the next, that of a boy of three years, the *occipital lobe* appeared to be the seat of the disease. The child took ill suddenly with vomiting and drowsiness, followed by a convulsion. When he regained consciousness two days later his neck was stiff, and, though he was quite intelligent and his fundus was normal, he was absolutely blind. He spoke and could stand, but could not walk. There was no weakness or inco-ordination of the limbs, and no ocular paresis. Sensation was unaffected and the knee-jerks normal; there was no ankle-clonus, and the plantar reflexes were flexor in type. A month later the child was beginning to see, and could walk with help; but about that time he began to have attacks of petit mal. Five months after the commencement of his illness he was quite well, with the exception of the petit mal.

Three cases with symptoms of *Polioencephalitis cerebelli* are recorded. In the first (girl of 4½), the attack followed whooping-cough, and began with fever and vomiting. Paresis of both legs and affection of speech followed, with slight affection of the sphincters. There was little headache and no loss of consciousness. Five or six weeks later she could just stand with legs wide apart, and tended to fall backward; on attempting to walk, the gait was wildly ataxic. The hands, especially the right, were feeble, and showed marked inco-ordination. When laughing, the left side of the face moved better than the right. Ocular movements good, except slight defect in upward movement of right eye; no nystagmus; no optic neuritis; pupils reacting well to light. Articulation somewhat bulbar in character, no aphasia, knee-jerks equal and active, ankle-clonus and plantar response doubtful. After admission the child rapidly recovered, and three months later walked quite well, showing no inco-ordination.

In the second case, that of a boy of fifteen months, the symptoms were unilateral. The child lost the power of the left side and developed rhythmic movements in the arm and leg, with marked intention tremor and inco-ordination, but no rigidity. He lost the power of walking; the knee-jerks were present and equal; there was no ankle-clonus, and the plantar reflexes were flexor. The eyes and eyesight were quite normal. A week later he could walk with help, and had only a slight intention tremor in the left arm. The mother noticed that he tended to hold his head with the left ear near the left shoulder.

A number of instances of *Polioencephalitis inferior*, with involvement of cranial nuclei, are given.

In the two first, the oculo-motor nuclei seemed involved. There was an acute onset of ophthalmoplegia, with loss of sight, followed by partial recovery, so far as the ocular muscles were concerned, but with permanent blindness. In four other cases

there was implication of the seventh, and in one of the eighth cranial nerve, which, judging by the clinical evidence alone, seemed to be due to encephalitis of the nuclei.

The writer hopes that if attention is drawn to the symptoms of polioencephalitis, it may result in further opportunities being obtained of investigating the pathology of the disease.

JOHN THOMSON.

**PSAMMOMATA OF THE DURA MATER.** (*Psammomi della dura* (58) *madre.*) ESPOSITO, *Manicomio*, Ann. xxxi., No. 2, 1905, p. 129.

CASE was that of a young girl who died *in status epilepticus* after long illness, in the course of which symptoms of cerebral tumour were present. At the autopsy the dura from without appeared to be absolutely normal. Its inner surface was studded with small tumours of varying size, many of which had undergone calcification. Microscopical examination showed *inter alia* the following points:—

Tumours appeared to be composed of a more or less dense fibrous tissue, with numerous lacunæ filled with calcified granules. At first sight they would have passed for calcified fibromata, but examination of two tumours, in which the calcareous process had not advanced so far, showed numerous cavities in the fibrous meshwork containing nests of small cells of irregular outline, with a large, central, granular nucleus, and occasional small fusiform cells, whose extremities were prolonged into the fine fibrils of the general stroma. The tumour was poorly provided with blood-vessels, without proper walls, being rather excavations in the neoplastic substance.

The tumour was attached to the tissues from which it arose by means of a fine peduncle, composed of fibrillar substance. The free surface of the dura, covering the internal face of the tumours, appeared to consist of cells of endothelial character, which were frequently found undergoing hyaline degeneration. The absence of any trace of inflammation in the adjacent dura mater at once negatives the idea that the tumours are products of inflammatory activity.

The author attaches great importance to vascular structure as a criterion of the sarcomatous nature of a tumour. The tumour which he observed differs from endotheliomata in the absence of large flat cells grouped together. Their fusiform cells were few in number, and the round cells have less of an endothelial character, while the stroma is much developed and the vascular supply is scanty.

Under the group of psammomata a number of widely differing

neoplasms have been included. The majority of writers regard psammomata as angiolithic sarcomata. The author would recognise three varieties: the endothelioma of the serous coat; the angiolithic sarcoma; and the third, of which this case is an example, the calcified fibro-sarcoma. He considers that the purely fibrous nature of the peduncle and absence of any trace of vascular origin distinguished these tumours from the growths described by Cornil and Ranvier as ampullary dilatation of vessel walls (similar to those normally found in the choroid), with a covering of flattened, calcified, epithelial cells, the tumour finally becoming pedunculated.

F. GOLLA.

**CONVULSIONS IN TYPHOID FEVER.** WILLIAM OSLER, *Practitioner*, Jan. 1906, p. 1.

THE occurrence of convulsions in the course of this fever is one which has received but little attention. Murdison, in a series of 2960 typhoids, noted convulsions in only six cases. Osler has notes of eight cases in a series of between 1500 and 1600. These cases he groups under three heads. In the first place, two cases had a history of the attack commencing with convulsions. No symptoms of any kind had been noticed previously. Secondly, in four cases the convulsions were apparently a manifestation of the toxæmia during the course of the disease. Thirdly, in the remaining two cases there were brain lesions to account for the fits, in one thrombosis of cerebral vessels, in the other tubercular meningitis. Dr Osler also mentions a case occurring in convalescence. Of the cases reported, only three died: one of a subsequent perforation, and two of the brain lesions mentioned above. The prognosis, then, is not very grave.

In some of these instances only one convulsion is reported. In others the convulsions were repeated at frequent intervals. The character of the convulsions is not described in all the cases. In some they were apparently epileptiform. Dr Osler's paper is a valuable addition to the literature of typhoid fever.

CLAUDE B. KER.

**MYOCLONUS MULTIPLEX: WITH REPORT OF A CASE.** HECHT, (60) *Am. Journ. Med. Sci.*, Dec. 1905, p. 1041.

THE writer refers to the great confusion that has arisen with regard to the nature of this disease, since Friedreich in 1881 first reported his case of "Paramyoclonus multiplex," and criticises the various theories that have been advanced to account for its patho-



genesis. He records in great detail a case which came under his own observation, and in which alcohol and chloral appeared to be the only two remedies capable of allaying the spasms.

JOHN D. COMBIE.

**ON A CASE OF TRAUMATIC HYSTERIA, WITH THE CLINICAL (61) PICTURE OF AN OPHTHALMOPLÉGIA EXTERNA.** (Ueber einen unter dem Bilde einer Ophthalmoplegia externa verlaufenden Fall von traumatischer Hysterie.) A. WESTPHAL (of Bonn), *Deutsche med. Wchnschr.*, June 1, 1905.

PATIENT was a miner, aged 46, who after an injury to the head developed the symptoms of a traumatic neurosis—headache, dizziness, quick heart action, vasomotor excitability, increase of mechanical muscular irritability, occasional tremor of the whole body. His mood was rather dull and hypochondriacal. When admitted to the psychiatric clinic, he appeared to present a bilateral complete ophthalmoplegia without implication of the levator palpebræ superioris or of the muscles of the iris. The paralysis of the eye-muscles was found to be variable, and under certain circumstances would partially or completely disappear; under certain circumstances, and especially when patient was examined by some new method, the eyeballs were rotated even to their normal extent. When, however, his attention was directed to the examination of the eye-muscles, he was unable to rotate the eyeballs at all. The eyeball would return from an excursion, not in an uninterrupted movement, but with a few halts, before reaching its normal position of rest. A tendency to pass into contracture was noticed in other muscles.

The variability of the paralysis under psychic influences led to the diagnosis of a functional disorder, simulating closely an organic ophthalmoplegia, such as is found sometimes in late apoplexy after head trauma, with hæmorrhage into the central grey matter.

C. MACFIE CAMPBELL.

**HYSTERICAL TIC.** (Le tic hystérique.) PITRES et CRUCHET, *Journ. de Neurol.*, Dec. 20, 1905, p. 541.

THE authors hold that tic may be a real clinical manifestation of hysteria, but exclude from the term certain tics which are merely added to, or are concomitant with, hysterical manifestations, also Meige's *rhythmic tics* of hysterical subjects.

Two cases are here recorded: one in a woman of 30, where the spasm, affecting the shoulder, neck, and face, followed on and

replaced neuralgias and convulsive crises. After lasting 8 months, the condition was cured in two sittings by hypnotism. A slight return  $3\frac{1}{2}$  months later, brought on by a dream, was entirely stopped by suggestion in the hypnotic state.

The second patient, a girl of 14, suffered from hysterical crises, with hiccough every day at certain hours, for a month; these disappeared, but tics of the shoulders, head, and face took their place, occurring at exactly the same hours each day, and persisted for 6 months in spite of all treatment. Pressure over one ovary could bring on the tic at other times. The condition ceased suddenly, coincident with the return of menstruation, but hemianæsthesia, which had been present throughout, persisted.

In both cases various actions (fixation of attention, respiratory exercises, etc.) which modify simple tic had no influence.

J. H. HARVEY PIRIE.

**HABIT SPASM IN CHILDREN.** GEORGE F. STILL, *Lancet*, Dec. 16, (63) 1905, p. 1754.

IN 100 consecutive cases of habit spasm, Dr Still found "screwing of the eyes" in 47, head-jerk in 30, affection of the upper limb in 22, and of the lower limb in 9. The production of sudden sounds was noted in 14 cases. The movements were usually strictly limited to one part at one time, but there was a striking tendency for one form of spasm to be replaced by another after a number of days, weeks, or months. Psychical disorders were present in some cases, usually associated with rather violent movement. Such disturbances might take the form of the utterance of foul language, apparently without reason and even unconsciously; of irritability of temper; or even of definite delusions or hallucinations.

As regards etiology, 53 cases were girls and 47 boys. The greatest incidence of the disease was between 6 and 8. Most of the children were of nervous temperament, and many suffered from other neuroses, such as talking in the sleep and enuresis. Most were sharp, responsive, and of quick intelligence. Rheumatism was of more than average frequency in the family history.

As regards local exciting causes, some definite irritation may be found not infrequently, and should always be looked for, because its removal, if possible, may stop the spasm. The age incidence suggests one source of irritation, namely, the outset of the second dentition. Conjunctivitis, hypermetropia, astigmatism, inflammation of the nasal mucous membrane, and perhaps worms may serve as exciting causes. Mental strain and mental excitement are important factors.

In diagnosis there is not usually much difficulty, but habit spasm may be mistaken for chorea.

As to treatment, a holiday in the country is often speedily followed by disappearance of the spasm, but there is a likelihood of recurrence if the child returns to school too soon. Lessons should be begun again gradually, and the subjects should be selected so as to avoid strain or worry. All forms of excitement and fatigue should be avoided. Late hours are particularly harmful. Any source of local irritation should be attended to, but operations (*e.g.* for adenoids) may make matters worse. Scolding or punishment can only do harm. Drugs are only of restricted value. Arsenic combined with bromide of potassium may do good. Ergot and valerian have also proved useful.

W. B. DRUMMOND.

**ON TWO CASES OF MYASTHENIA GRAVIS.** J. MITCHELL CLARKE,  
(64) *Bristol Med. Journ.*, Dec. 1905, p. 308.

THE author reports two fatal cases :—

The first case was that of a single woman, aged 32, who died from exhaustion and cardiac failure fourteen months after the appearance of nasal articulation, which was the first symptom of the disease. For eighteen days prior to death, dysphagia was so great she had to be fed by nutrient enemata, œsophageal feeding having to be abandoned because of the distress caused by the passage of the tube.

The second case was that of a woman, aged 63, who died six weeks after the first symptom appeared "in an access of dyspnœa."

In neither case, unfortunately, was there a post-mortem.

The pathological observations of Laquer and Weigert, Goldflam, Link, Hödelmoser, and Hun are briefly referred to.

The administration of thyroid extract and the hypodermic injection of strychnine were accompanied by improvement in the first case.

EDWIN BRAMWELL.

**ON THE DISTURBANCES OF MOVEMENT IN INFANTILE**  
(65) **CEREBRAL HEMIPLEGIA, AND ON DOUBLE ATHETOSIS.**

(Über die Bewegungsstörungen der infantilen cerebralen Hemiplegie und über die "Athétose double.") LEWANDOWSKY,  
*Deutsch. Zeit. f. Nervenheilk.*, H. 5-6, 1905, p. 339.

THE writer draws a distinction between the contracture of infantile and of adult hemiplegia. In the latter case the contraction of the

muscles can be overcome; they can be stretched, and have a certain elasticity. In the former the contracture is "fixed"; it is one of connective tissue rather than of muscle, and its elasticity is of a different quality. While defect of movement in hemiplegia is often due to incomplete or complete loss of inhibitory control over the antagonists, in infantile cases it would appear that some muscles and their antagonists function normally as a group, other muscles and their antagonists fail together—an important distinction. For instance, it is common to find both internal and external rotation of the arm faulty, both supination and pronation weak or absent, while flexion and extension at the wrist may both be strong, etc.

In explaining what is meant by athetosis, the author makes an elaborate differentiation between athetosis and "chorea" of a hemiplegic nature. In athetosis there is always a co-existing hemiplegic paralysis of some kind, whereas chorea occurs with no disturbance of voluntary movement on that side. Athetosis is revealed in the attempt at voluntary movement, and it is an intermittent spasm; whereas voluntary movement does not of necessity elicit the chorea. An athetotic spasm may be of some duration. A patient whose hand is passively closed may be unable to open it for a space. Athetosis is specially characteristic of infantile hemiplegia, and is to be explained as a disorder of function, and not of structure. By "pseudo-athetosis" is signified the involuntary movements—*e.g.* of fingers and arm—that occur on the hemiplegic side when a patient is exercising other muscles, as, for instance, in walking. When the hemiplegic arm is tested, the patient being at rest, well-marked association movements are seen in it; but when he is walking, movements more of an athetotic nature are exhibited.

Four cases of double athetosis are detailed. Double athetosis is not equivalent to a double hemi-athetosis, but ought to be used to indicate a special relation between individual parts of the body and limbs. Movement of one limb entails and is inevitably succeeded by movement of another. Double athetosis is a generalised association of such movements. The patient may sit for hours motionless, but when the time comes for him to go to bed, or when everyone else in the ward is getting into bed, the psychological moment has arrived, and his first movement is at once followed by a whole series.

There is little reference to pathological data.

S. A. K. WILSON.

**ON THE COURSE OF THE RETROGRESSION OF HEMIANOPIC  
(66) DISORDERS AFTER PARALYTIC ATTACKS.** (Ueber den  
Gang der Rückbildung hemianopischer Störungen nach para-  
lytischen Anfällen.) A. PICK (of Prague), *Deutsche med. Wchschr.*,  
Sept. 28, 1905.

PICK calls attention to a phenomenon which he has observed after a paralytic attack in cases of general paralysis during the period of disappearance of a typical homonymous hemianopia. To take the case of a left-sided hemianopia. When an object was carried through the normal field (right) into the hemianopic field, patient only followed the object as far as the middle line; but when the object was brought from the left side into the outer limit of the hemianopic field, the patient immediately looked in that direction, sometimes regularly, sometimes only occasionally. There are two ways in which the phenomenon may be explained. The fixation may be considered as due to an act of attention, and for this to take place, a certain activity of function of the cortico-retinal apparatus is necessary. The return of this function is shown by the reaction to the object brought into the periphery of the visual field. As the object is carried from the periphery of the hemianopic field inwards, the patient follows it for a short space, but soon loses it; the distance increases as the hemianopia improves. The hemianopia, therefore, seems to clear up from the periphery inwards. The importance of the factor of attention is seen by the fact that, while the patient reacts to an object in the periphery of the hemianopic field, on the other hand, when another object is simultaneously brought into the periphery of the normal field, the patient invariably fixates the latter object (? because better seen).

Another explanation of the reaction to an object in the hemianopic field may be found in the hypothesis that the movements of fixation, at first completely dependent upon the visual centre, become in time more automatic, and are handed over to a lower reflex arc, which passes directly from the primary optic centres to the centres for the eye-muscles. The phenomenon under discussion would be then due to the dissociation of voluntary and involuntary fixation, the latter being carried out by this reflex arc. That involuntary fixation returns before voluntary fixation has been demonstrated after operation on the congenitally blind.

It is legitimate to assume that in the early stage of restoration of the hemianopic field, involuntary fixation first returns, and that the return of the function of the cortico-retinal apparatus proceeds from the periphery inwards.

C. MACFIE CAMPBELL.

**PSYCHICAL AND SENSORY REACTION OF THE PUPILS.**

(67) (Über die psychische und sensible Reaktion der Pupillen.)

HÜBNER, *Centralbl. f. Nervenl. u. Psych.*, Dec. 15, 1905, p. 945.

THE author examined the condition of these pupillary reactions in cases of imbecility and dementia præcox, and found them absent in 75 per cent. of the latter disease, 51 examples being taken. He noted their absence, however, in 3 normal women over 48 years of age, in whom the normal light reflex was present. In dementia præcox, both reactions are met with in only 14·3 per cent. of cases, whereas in all other functional psychoses there is no impairment of either. In imbeciles and idiots a pupillary response to psychical and sensory stimuli was rarely wanting. As far as his experience goes, the author has never seen a return of these reflexes, although the mental condition of some patients has undergone a considerable change since it first came under notice.

S. A. K. WILSON.

**THE SENSE OF PAIN.** (*Le sens de la douleur.*) IOTAYKO, *Journal de Neurologie*, oct. 5 et 20, 1905.

Mlle. IOTAYKO discusses in this paper the views that are at present held in regard to the sensation of pain. Starting with the consideration of the production of pain, she accepts the statement of Richet that it is due to a powerful stimulation of nerve endings. This is illustrated by the results obtained from the application of pressure on the back of the hand, when by gradually increasing the pressure three stages are passed through—a stage of non-perception, of perception without pain, and of perception with pain. By the use of the Algèsimetre of Chéron (an instrument by which the exact amount of pressure that results in pain is measured), Iotayko and Stefanowska have found a variation in the perception of pain in different individuals. In those persons who are less susceptible to pain, "pain spots" are less numerous than in the more sensitive individuals. Different parts of the skin vary in their sensitiveness. Thus of five regions selected, the region of the temple is most sensitive; then follows the anterior surface of the forearm, the pulp of the ring finger, back of the hand, and the pulp of the middle finger. Contrary to what is found in regard to touch, the pulp of the fingers is not highly endowed with sensibility to pain. Attempts have been made by numerous writers to differentiate the different kinds of pain, Hahnemann distinguishing as many as 73. There are, however, points which are sensitive only to certain sensations, the points for temperature, pain, and

touch being anatomically distinct. Frey's scale of the sensitiveness of different regions shows a wide difference in the sensibility of the different parts, from the exquisite sensitiveness of the cornea, to the comparative insensitiveness of the sole of the foot. In regard to the histological localisation of the four cutaneous senses, Frey's views are quoted. According to him the corpuscles of Meissner respond to pressure; pain excites directly the free nervous terminations that are found in the cornea; while in the skin these free terminations are situated more superficially than the organs for pressure. The corpuscles of Kranze serve the sensation of cold, while the corpuscles of Ruffini serve that of heat. In regard to the mechanism by which pain reaches the brain, the different and conflicting views are briefly stated, as also those in regard to the localisation of the centre in the brain in which the pain is perceived. No view has yet been expressed that can be accepted with certainty. In the elucidation of this part of the subject some interesting observations are quoted concerning the results of the artificial production of analgesia by chloroform, cocaine, menthol, etc. From some careful experiments by Ioteyko and Stefanowska, it is shown that the left side of the body is more sensitive to pain than the right. In the chapter dealing with the remote effects produced by pain, some interesting results are brought together. The effect of pain on temperature is to lower it. The effect upon the heart is variable, arresting it, slowing it, and in some instances increasing its rate. The effect of pain upon respiration is also varied, sometimes slowing the rate, while it becomes more profound and irregular according to L. Meyer; while Montegazza found a diminution in the exhalation of carbonic acid. Pain retards digestion. The facial and other expressions produced by pain have been a subject of study by numerous observers, and references are given here to the most important of these. The sensibility to pain according to sex, age, race, etc., is discussed. According to Montegazza, the circumstances that increase the sensibility to pain are a refined sensibility, high intellectual acquirements, a high degree of civilisation, the feminine sex, youth, certain degrees of heat, the abuse of coffee, the sudden transition from pleasant sensations to painful. The discussion of such questions as the radiation of pain is treated very imperfectly, and many of the most instructive data are ignored. Thus "*La douleur est sentie en cercle, et souvent aussi on ressent des élancements douloureux. L'irridation de la douleur s'observe aussi dans les maladies (névralgie dentaire, coliques hépatiques, coliques néphrétiques)*" is practically all that is said of a very important part of the subject—a part, indeed, whose proper appreciation affords the key for the solution of many questions that are left obscure in this article. The article concludes with a very

good bibliography, but some very important papers are not included. In fact some of the best work, and certainly some of the most original work, has been done by British investigators, but evidently the authoress is not aware of the far-reaching results arrived at by such observers as Ross, Sherrington, and Head.

JAMES MACKENZIE.

# **DISTURBANCES OF SENSATION IN ACUTE LOCALISED**

- (69) **ISCHÆMIA.** (Über Sensibilitätsstörungen bei akuter lokaler Ischämie.) SCHLESINGER, *Deutsch. Zeit. f. Nervenheilk.*, H. 5-6, 1905, p. 375.

THE author has been able to examine some cases of cardiac disease in which blocking of a peripheral vessel has occurred. If such a case be seen immediately after the circulation is thus locally cut off, it will be found that there is loss of all forms of sensibility, deep and cutaneous, beyond the line of the blocking, and more or less corresponding to the distribution of the affected vessel. The motor paralysis that occurs is not detailed by the writer, who remarks on the great physiological interest of this sudden loss both of superficial and of deep sensation, and their equally sudden return after the circulation is restored. The explanation seems to rest in the functional disturbance of nerve endings caused by the acute ischæmia.

S. A. K. WILSON.

# **BABINSKI'S SIGN IN SCARLET FEVER.** (La signe de Babinski

- (70) dans la scarlatine.) KIROFF (Soc. de Neurol.), *Rev. Neurolog.*, Nov. 30, 1905, p. 1119.

KIROFF found Babinski's sign on one or both sides in seventeen cases of scarlet fever, ten of which died. The patients' ages ranged from 2 to 27. With the subsidence of acute symptoms the reflex became normal. In most of the cases the knee-jerks were either lost or diminished. In some of the fatal cases the sign, which at first could be easily obtained, became difficult to elicit or entirely disappeared shortly before death. The phenomenon is attributed to perturbation of the pyramidal system, which is affected by the scarlatinal toxins like the heart, the kidneys, and the meninges. This hypothesis is supported by the same sign having been found by Kiroff in some severe cases of diphtheria. In further confirmation of this view, the reviewer can cite four cases of enteric fever lately under his care in which Babinski's sign was obtained. The phenomenon was transient, and was associated with no other sign of disease of the pyramidal system.

J. D. ROLLESTON.



**THE "EXTENSOR PHENOMENON."** (Das "Streckphänomen.")  
(71) SAXL, *Neurolog. Centralbl.*, Dec. 16, 1905, p. 1140.

A HEMIPLEGIC girl with flexed hand and wrist, which she was incapable of extending voluntarily. She could always do so if she extended her elbow. The condition seems to be analogous to the tibialis phenomenon of Strumpell. S. A. K. WILSON.

**WORD-BLINDNESS, WITH THE RECORD OF A CASE DUE TO A**  
(72) **LESION IN THE RIGHT CEREBRAL HEMISPHERE IN A**  
**RIGHT-HANDED MAN; WITH SOME DISCUSSION OF THE**  
**TREATMENT OF VISUAL APHASIA.** CHARLES B. MILLS  
and T. H. WEISENBERG, *Medicine*, Nov. 1905.

THE interesting case here reported is that of a right-handed man—he ate, wrote, and did by preference almost everything with his right hand, and was not ambidextrous—who, after a severe apoplectic attack, supposed to be due to a cerebral hæmorrhage, suffered from left-sided hemiplegia and aphasia. The hemiplegia improved considerably, but the aphasia, consisting principally in word- and letter-blindness, persisted. The authors hold that the speech defect was due to a lesion in the right side of the brain. Instances of "crossed aphasia" are very rare. Senator in 1904 was able to collect only eleven cases from the literature.

When the patient was first seen by the authors, a year after the apoplexy, he was only able to recognise six letters, while after six weeks' training he was able to recognise nearly all the letters of the alphabet. The method adopted was to show the patient a letter or word, make him pronounce it, pick it out from among other letters or words and try and picture it.

EDWIN BRAMWELL.

**THE CONDITION OF THE FUNCTIONS OF THE BLADDER IN**  
(73) **CEREBRAL HEMIPLEGIA.** (Ueber das Verhalten der  
Blasenthätigkeit bei cerebraler Hemiplegie.) ED. MÜLLER,  
*Neurolog. Centralbl.*, Dec. 1, 1905, p. 1101.

It is commonly taught in the text-books that unilateral brain lesions are not, as a rule, accompanied by bladder trouble, apart from the familiar incontinence during an apoplectic attack, where the bladder symptom is ascribed to mental dulness. Bilateral brain lesions, on the other hand, are more frequently accompanied by bladder trouble.

Müller's paper claims to show that bladder troubles are a very frequent accompaniment of ordinary hemiplegia from vascular lesions, even in the chronic stage, when there is no longer any question of mental blunting and where sources of fallacy, such as prostatic enlargement, have been excluded. The disorders of micturition, however, are slight in degree and varying in intensity. So much so, that they have to be enquired for by the physician, inasmuch as the patient rarely mentions them spontaneously. A moderate degree of "imperative incontinence" is the rule, a precipitancy which makes the patient hurry to micturate, to prevent involuntary emptying of the bladder. The degree of bladder affection is precisely similar to that which occurs so constantly in disseminated sclerosis.

Similar slight bladder troubles are often noted amongst the earliest phenomena of cerebral tumours, even where the intellectual functions are unaffected.

PURVES STEWART.

**INTERMITTENT CLAUDICATION.** (Zur Kasuistik der intermittierenden angiosclerotischen Bewegungsstörungen [Dysbasie, Dyskinesie] des Menschen.) W. ERB, *Deutsche Zeit. f. Nervenheilk.*, Bd. 29, 1905, p. 465.

THE patient, a man of Jewish race, 32 years of age, who smoked 12 to 15 cigarettes daily, had syphilis three years previously, for which he underwent three courses of mercurial inunction at intervals of about a year.

A year before he came under observation, he began to have a "hot, tired feeling" in the left leg on exertion. This steadily increased, so that it occurred after slighter and slighter exertion. The left thigh became affected some time later. On examination, both femoral and popliteal pulses were equal on the two sides, but in the left foot neither artery nor pulse could be felt. Under mercury, iodides, galvanism, hot foot-baths and rest, the patient improved.

Some three years later, the patient again came under observation. By this time, the right leg had begun to show symptoms similar to those in the left, *i.e.* pain on walking, which passed off on resting. But, in addition, he now complained of pains in the left upper arm, along its inner side, and frequent tingling in the left finger-tips. The left radial pulse was much feebler than the right. The pulse was absent in the left foot, that in the right foot was much weaker than before.

The occurrence of symptoms in the upper limb, precisely similar to those in the lower limb, leads Erb to classify the case under the wider category of "dyskinesia angio-sclerotica," rather

than the more limited one of "intermittierendes Hinken." In this particular case, it is probable that syphilitic arterio-sclerosis played a part, in addition to excess in tobacco. Yet anti-syphilitic treatment failed to arrest the disease.

PURVES STEWART.

**HÆMATEMESIS IN ORGANIC NERVOUS DISEASES (TABES).**

(75) (Haematemesis bei organischen Nervenerkrankungen [Tabes].)

ALFRED NEUMANN, *Deutsch. Zeitschr. f. Nervenheilk.*, Bd. 29, 1905, p. 398.

THE author describes a case of tabes with gastric crises and frequent attacks of hæmatemesis. The vomit usually consisted of bile-stained fluid with some blood, but occasionally almost pure blood was brought up. The patient was under observation for nearly six months. The blood-pressure rose from almost normal to 160 or 170 mm. of Hg. during the crises, and always to over 200 mm. during the act of vomiting. The bleeding was attributed to the sudden rise in pressure rupturing larger or smaller vessels in the gastric mucous membrane—all complications such as ulcer, cancer, etc., being fairly well excluded. According to Pal, this rise in blood-pressure is a constant feature of the gastric crises in tabes; but in 105 cases culled from literature by the author, only 10 were found with hæmatemesis. Short digests of these are given; only in 2 was the symptom a marked one.

J. H. HARVEY PIRIE.

**A CONTRIBUTION TO THE CLINICAL STUDY OF THE**

(76) **PHARYNGEAL REFLEX.** (Contributo allo studio clinico del riflesso faringeo.) FORLI e GUIDI, *Ann. dell' Instit. Psichiat. di Roma*, Vol. iv., 1905, p. 75.

THE pharyngeal reflex is essentially a movement of elevation and constriction of the pharynx, and of elevation of the soft palate.

The authors discuss results of various experiments and views of writers as to the motor supply of the pharynx, and the part taken by the glosso-pharyngeal nerve in conduction of tactile and sensory impulses; the majority of physiologists concur that the sensory function of the ninth nerve is exclusively gustatory. The sensory functions of the pharynx may be said to be supplied by the vagus, while its sensorial functions are supplied by the glosso-pharyngeal nerve.

The pharyngeal reflex, however, is by no means a constant phenomenon. The authors examined 98 cases which presented no nervous lesion. Of these, 63 were above fifty years of age, and

35 below. In those below fifty, the pharyngeal reflex was brisk in 42 per cent., weak in 14 per cent., scarcely perceptible in 20 per cent., and absent in 15 per cent. In those above fifty it was brisk in 40 per cent., weak in 8 per cent., scarcely perceptible in 20 per cent., and absent in 31 per cent.

The pharyngeal reflex was next examined in numerous hysterical cases. Kattwinkel has recently examined 104 hysterical cases, and found that the absence of the pharyngeal reflex bears no relation to the presence of thermal and pain sensibility, and hence is to be regarded as an interruption of a cerebral reflex path.

The authors found that in 58 hysterical cases the reflex was brisk in 13 per cent., weak in 13 per cent., scarcely perceptible in 10 per cent., and absent in 65 per cent. In only one case was there tactile anæsthesia of the pharynx, and in this, as in all the others, pain sensation was present. On the other hand, gustatory sensation was absent or impaired in a large number of cases, and this is thought to explain the results of Volkman. This author found that, after section of the glosso-pharyngeal, the pharyngeal reflex was abolished; whilst after section of the trigeminal, which indubitably supplies sensory fibres to the isthmus of the fauces, the pharyngeal reflex persisted.

Kattwinkel and Marie considered that in cases of hemiplegia the pharyngeal reflex was abolished in 50 per cent. of cases of right hemiplegia and diminished in 20 per cent., whilst in cases of left hemiplegia it was absent in 4 per cent., and diminished in 20 per cent. Hence they draw the conclusion that the seat of the reflex is probably in the right corpus striatum.

The authors examined the reflex in 12 cases of right hemiplegia and 10 of left. They conclude that in the majority of cases of hemiplegia the reflex obtained by stimulation of the pharynx on the side corresponding to the hemiplegia is less brisk than that obtained from the opposite side, and that this is independent of any appreciable diminution of sensibility. The side affected by hemiplegia did not appear to have any influence on the results obtained.

F. GOLLA.

#### **THE RÔLE PLAYED BY INTENSE COLD IN THE PATHOGENY**

(77) **OF ACROPATHIES.** (*Rôle du froid intense dans la pathogénie des acropathies.*) ÉTIENNE, *Arch. gén. de Méd.*, Dec. 20, 1905, p. 3265.

In the first case the patient was exposed a whole night to intense cold, and frost-bite supervened, associated with progressive and uninterrupted sensory phenomena, ending in symmetrical mutilation of the extremities.

In the second, a mechanician noticed on a particularly cold morning that his uncovered hands had become numb, cyanotic, and locally asphyxiated. Spasm of the arterioles and dilatation of the venules were responsible for these symptoms, which disappeared in summer and returned the next winter. Under the influence of the spasmodic contractions the radial arteries responded by local endarteritis obliterans, the result being diminution in the radial pulse.

In the third case the guard of a snowed-up train was forced to make a journey of about a mile through chest-deep snow, and a typical polyneuritis was the result. The transitory arterial spasms occasioned by the cold so affected the vessels that eventually a local arterio-sclerosis made its appearance. Under a slightly more heavy muscular strain than usual a prolonged and painful arterial spasm occurred, and in the morning the fingers were already black.

Evidently, then, cold may act directly or indirectly, in the latter case devitalising a part by arterial spasm, prolonged or repeated, setting up an arterio-sclerosis, and so making local death possible, insidiously or suddenly.

S. A. K. WILSON.

### PSYCHIATRY.

**A CLINICAL, ANATOMICAL, AND PATHOLOGICAL CONCEPTION OF THE FORMS OF IDIOCY WHICH ARE THE RESULT OF MENINGITIS.** (*Concetto clinico, anatomico, e patologico, delle idiozie meningitiche.*) G. B. PELIZZI, *Riv. Sper. di Fren.*, Vol. xxxi., F. 3-4.

THE symptom "idiocy," in its various grades, finds its pathogenesis either in a chronic and hereditary intoxication of the nervous system, which may have acted through some generations, or in a common inflammatory process of the brain and its membranes. The degenerative element, however, is not without some influence on the inflammatory forms.

In the toxic forms the poison acts so as to produce an arrest, a retardation, or an altered development of the brain without there being any recognisable pathological process; in the inflammatory cases it has led to a diminished resistance, which renders the brain and its membranes exceedingly susceptible to infections and ordinary pathological processes. It is this special vulnerability which constitutes the hereditary degenerative substratum of the meningitic idiocy. These processes may start at any period from the earliest embryonal stages to puberty, and according to the epoch at which they arise, to their extent, and to their intensity, they will produce results which may be grouped as follows: (a) pseudo-

hypertrophic forms ; (b) forms with simple and uniform atrophy ; (c) forms in which the brain tissue has been destroyed in scattered foci.

But it is impossible to draw any sharp lines between these three groups, as it is also between toxic and inflammatory idiocy.

The most important differential characters, from the clinical point of view, are the motor symptoms. In the pseudo-hypertrophic forms the motor symptoms consist of general convulsions of an epileptic type ; in the atrophic group, of generally diminished muscular tone ; in the third group, of paralytic lesions, almost always spastic and focal.

The anatomical changes found in the first group consist of an increase in the volume of the whole brain or of some parts of it ; histologically, there is a great proliferation of the neuroglia, and the nerve cells are atypical and embryonal.

In the second group the brain is uniformly affected ; its volume is diminished, the convolutions are flattened, but the surface is smooth and the sulci are regular. Microscopically there is diminution in number and volume of the nervous elements, and slight overgrowth of the neuroglia.

In the destructive forms, scattered cicatricial contractions, erosions, microgyria, and porencephaly are the prominent characters. Microscopically, the nerve elements are found to have almost entirely disappeared in the area affected, and in the other portions of the brain the changes resemble those of the previous class.

The atrophic group includes the cases of meningitic idiocy, properly so called, of microcephalic idiocy, and of hydrocephalic idiocy. It is with these three that the author deals in this paper.

In the case of the meningitis leading to idiocy, we must assume that the inflammatory process has spread from the meninges to the cerebral cortex, and, depending on the extent to which this has taken place, shall we find a condition of simple meningitis or meningo-encephalitis. In the latter affection the inflammatory process penetrates deeply into the brain substance, and considerable portions of the brain may be destroyed by it. But in cases of simple meningitis, on the other hand, the evidences of injury may be almost wanting, only a slight thickening of the membranes being visible, while the cortex shows no microscopical signs whatever. Every degree of injury is met with in the brains of the meningitic idiots, and their pathological anatomy borders on a microcephalic condition on the one hand, and on meningo-encephalitis on the other hand. Generally the pia is thickened, but strips from the cortex without tearing it. Sometimes the volume of the convolutions is normal, but, at other times, they are small, and this diminution may reach the degree of a true microcephalic brain. The lateral ventricles are often dilated. The histological

examination shows a slight overgrowth of the neuroglia in the outer layer, a diminished number of small and medium pyramidal cells, very poor development of the capillary network in the cortex, and thickening of the adventitia of the vessels. These results are probably produced by a serous meningitis which interferes with the nutrition of the nervous structures, and the changes mentioned are the result. Such a simple meningitis may be started by any of the ordinary invading organisms. There can be no doubt, however, that the different powers of resistance of the subjects, the neuropathic predispositions, the age of onset, must be important factors in the determination of the degree of these changes.

Dealing with the pathogenesis of microcephaly, the author shows that the degrees and variations of the brain in this condition may be as marked as in all the other classes of idiocy which are due to a meningitic process. The brain may be uniformly diminished in size, or some convolutions may be small and others very much larger than normal, even to the extent of macrogyria. The two hemispheres may be unequally affected. There is no type of brain which is characteristic of these cases, such as there would be if the condition were an entity outside ordinary pathological processes. According to the author, they can be explained only by admitting some inflammatory pathological process, and that process, in his opinion, is a serous meningitis.

With regard to those cases in which, together with a small brain, there is evidence of coarse destructive lesions, it is suggested that in these, as in all other forms, there has been an inflammatory process which has diffused from some points of greater intensity, and that it is at these points of intensity that the loss of substance has occurred.

Turning next to the pathogenesis of hydrocephalus, the author first shows that hydrocephalus may be internal or external, active or passive, true or false. The internal, active, true hydrocephalus may be acute or chronic, congenital or acquired. It may occur as a simple condition, with ventricular dilation and with characteristic alterations of the choroid plexuses and the ependyma, or it may be associated with macroscopical destructive sclerosis of the cerebral grey substance, or of the ventricular parietes, with external meningo-encephalitis and coarse cortical lesions, with simple external meningitis, and with rickets. Most frequently it is found with the simple meningitis. The dilatation of the ventricles must be due to an increased secretion, or to a diminished absorption.

The examination of recent cases points to an inflammatory origin, as proved by the diffusely or locally thickened ependyma, with smooth or nodular surface, and by the thickened and adherent choroid plexuses.

Most cases start acutely with grave cerebral symptoms; then follow convulsive attacks. Later, the effects of mechanical pressure appear. The author quotes a case which showed evidence of an internal, ventricular meningitis, certainly inflammatory, but with a purely serous exudation. Most authorities now agree in attributing the condition to an acute infection, probably of gastro-enteric origin, which gives rise to a serous meningitis.

Syphilis is also mentioned as possessing a greater importance in the production of hydrocephalus than of any other form of idiocy, but that is not to say that it is a factor in every case.

With regard to the relation which exists between rickets and hydrocephalus, the author holds that rickets cannot directly be the cause of that condition. But in rickets the general nutrition of the body is so generally interfered with that the invasion by organisms is greatly facilitated.

This condition of hydrocephalus is the result of an inflammatory process of varying nature, which gives rise to functional and anatomical changes in the tela choroidea, the choroid plexuses, and the ependyma. But beside these cases associated with simple meningitis, there are others which show signs of a cerebral sclerosis or atrophy, combined with a true hydrocephalus. The patients exhibit all the characters of infantile spastic hemiplegia.

In these, the sclerosis has affected the nuclei at the base of the brain, and has spread into the hemispheres. The ventricle on that side on which the nuclei have been attacked is the first to be dilated, and then the inflammatory process invades the ependyma and internal meninges on the opposite side and produces a similar result. Then again, there may be a hydrocephalus associated with a meningo-encephalitis, in which portions of brain substance in various situations have been destroyed.

It will be seen, therefore, that intermediate grades between these three groups are common. Finally, the author admits that although there may be some lacunæ in the chain of facts, there exist so many clinical, anatomical, and pathological resemblances between these forms of idiocy as to justify their being grouped together under the term "meningitic idiocy."

R. G. Rows.

**THE DEPRESSIONS OF ADVANCED LIFE.** (Die Depressions-  
(79) zustände des höheren Lebensalters.) R. GAUPP (of Munich),  
*Münch. med. Wchnschr.*, Aug. 8, 1905.

GAUPP gives in a brief manner the results of his analysis of all the cases of depression occurring in patients over the age of 45 who were admitted to the Heidelberg clinic within a period of ten years. His work was originally intended to form part of a wider



scheme started in the same clinic to study the influence of advanced life upon mental diseases. The results here given are based on a material of 300 cases of depression, and 51 manic cases who had also to be included in view of the intimate relationship between certain forms of depression and manic attacks.

According to the statistics obtained, simple and periodic mania is more frequent in men than in women ; manic-depressive insanity, with equal development of both phases, is equally common in men and women ; manic-depressive insanity, with preponderance of the depressed form, is more common in women than in men.

The course of the cases of manic-depressive insanity was very varied, the following varieties being seen : one single manic attack, several manic attacks, several manic attacks with transitory depression, depressive attacks with rare manic phases, several depressive attacks, one single attack of depression of this type. Sometimes the manic attacks tended to become more severe in advanced life. Acute periodic mania (*i.e.* one form of manic-depressive insanity) frequently passed into a hypomanic condition with mental enfeeblement. It is an open question whether the latter is not due to associated senile or arterio-sclerotic changes. "Mixed states" of manic-depressive insanity (Kraepelin) frequently simulated mental deterioration, which the course of the disease showed, however, to be absent. Circular cases were found sometimes to continue to present in advanced life an alternation of the same clean-cut pictures, but frequently these became rather toned down.

In some cases, where the depressive phases were the more marked, the feeling of subjective insufficiency and retardation were absent ; in one case a state of anxious agitation represented the depressive phase.

Twenty-nine women and twelve men presented recurrent attacks of depression without any manic phase. In one group the attacks were characterised by psycho-motor retardation and a feeling of insufficiency, and terminated in recovery. These cases belong, according to Gaupp, to manic-depressive insanity. In a second group of recurrent depressions the patients were hypochondriacal, anxious, voluble in complaint. The onset was gradual, the outcome uncertain. Sometimes the patients recovered, sometimes the depression persisted, and in one case mental deterioration set in after the second attack. This group is more akin to Kraepelin's melancholia of the involution period, but the attacks are usually less severe, of shorter duration, and more amenable to external influences.

Twenty-six women and twenty-two men presented the picture of Kraepelin's melancholia. The question of the nosological position of this type is complicated by the following facts : Patients

may have recurrent attacks of melancholia, and in the intervals show no defect symptoms; in cases of periodic depression with onset before the involution period, the later and more severe attacks may present the symptoms and clinical course of melancholia.

Gaupp divides his cases of depression arising on an arterio-sclerotic basis into seven groups: 1. Chronic depressive dementia, with death after several apoplectic attacks. 2. A variety of symptomatological pictures following directly an apoplectic attack—among others an arterio-sclerotic Dämmerzustand. 3. Progressive development of neurological symptoms (especially aphasic), accompanied by a paranoid or hypochondriacal trend, and sometimes with anxious delirious episodes. 4. Cases running a quicker course, *e.g.* delirious confusion following a hypochondriacal stage, and passing into dementia. 5. Several cases presented a series of attacks (*e.g.* aphasic) before the development of mental symptoms, which consisted chiefly in an agitated depression with self-accusations. 6. In other cases depression preceded the neurological symptoms, and it was not easy to say whether the initial depression was really the expression of an arterio-sclerotic process, and not an independent condition, complicated later by arterio-sclerosis. 7. Cases with arterio-sclerosis of the coronary arteries presented episodes of anxiety referred to the heart region, with a permanent hypochondriacal mood.

Senile depressions varied both in symptoms and in course; the mood was usually anxious and hypochondriacal, and the symptom-complex of a cheerful presbyophrenia was found to be only one stage in a senile psychosis, and to be usually preceded by anxious or hypochondriacal periods.

There were thirty-nine cases of atypical depressions, some of which might be looked on as individual variations due to constitutional peculiarity; of this nature were cases described by some authors as "hystero-melancholia," "hystero-hypochondria." Of more importance was a group of patients who between 50 and 60 developed, after a short period of hypochondriacal depression, an acute anxiety psychosis with hallucinations and delusions, confusion and disorientation, resistiveness, and which frequently terminated in death through exhaustion, but in some cases passed into a condition of apathetic dementia, with stereotyped residuals of emotional gestures.

Gaupp insists on the necessity of keeping these cases separate from cases of late catatonia; and he isolates another group of patients who, after an hallucinatory excitement with anxiety, pass into an emotionally indifferent dementia, with variable ideas of persecution. These terminal dementias call for further analysis.

He next describes, under the head of "Depressive Climacteric

Excitement with Outcome in Mental Enfeeblement," a small group of female patients between 45 and 60 who, after a preliminary change of mood and general nervousness, developed excited lamentations, self-accusations, worry for the future, and terrifying hallucinations, but without clouding of consciousness; during the excitement there was noted loquacity, distractibility, and flight of ideas; the course was remittent. Recovery never took place, although the whole condition gradually simmered down.

The work done by Gaupp must prove a stimulus to the further study and differentiation of the large groups which have been established largely under the influence of the Heidelberg school.

C. MACFIE CAMPBELL.

---

## Review

**DIE ERKRANKUNGEN DES RÜCKENMARKES UND DER MEDULLA OBLONGATA. III. Medulla oblongata.** Prof. Dr E. v. LEYDEN and Prof. Dr A. GOLDSCHIEDER, Berlin. Second Edition. Vienna: Alfred Hölder. Pp. 84. M. 2.50.

THIS work contains six chapters, dealing respectively with progressive amyotrophic bulbar paralysis, acute bulbar paralysis, bulbar paralysis without anatomical change (*myasthenia gravis*), pseudo-bulbar paralysis, ophthalmoplegia (chronic progressive, and acute), and recurring oculo-motor palsy (*migraine ophthalmique*).

Diseases of the medulla are so varied in their symptoms and so important from a prognostic point of view that the authors' plan of dealing with them in a special section or volume is to be highly commended. The work cannot fail to be of real value. The authors give, within narrow compass, a very readable, wonderfully full, and generally lucid account of all that is known regarding these affections of the medulla. In some parts, we think, the arrangement of material might be clearer, notably in the chapter on ophthalmoplegia, and the addition of more illustrations and of references to literature would greatly enhance the value of the work. The letterpress is excellent.

A. W. M.

# Bibliography

## ANATOMY

- EDINGER und WALLENBERG. Bericht über die Leistungen auf dem Gebiete der Anatomie des Centralnervensystems in den Jahren 1903 und 1904. Hirzel, Leipzig, 1905, M. 4.
- VAN GEHUCHTEN. Le faisceau en crochet de Russell ou faisceau cérébello-bulbaire. *Névrose*, Vol. vii., f. 2, 1905, p. 117.
- KRAUSE. Die Endigung des Nervus acusticus in Gehörorgan des Flussneunauges. Reimer, Berlin, 1905, M. 1.
- BACH. Ist die Kreuzung des Trochlearis eine totale oder partielle? *Centralbl. f. Nervenheilk u. Psychiat.*, Jan. 1906, S. 16.
- RAMON y CAJAL. Vergleichende Strukturbeschreibung und Histogenesis der Hirnrinde. Anatomisch-physiologische Betrachtungen über das Gehirn. Barth, Leipzig, 1906, M. 6.
- O. CHARNOCK BRADLEY. On the Development of the Hind-Brain of the Pig. *Journ. of Anat. and Phys.*, Vol. xl., Part ii., p. 133.
- GEMELLI. Sur la structure des plaques motrices chez les reptiles. *Névrose*, Vol. vii., f. 2, 1905, p. 105.
- PAUL FLECHSIG. Einige Bemerkungen über die Untersuchungsmethoden der Grosshirnrinde insbesondere des Menschen. *Arch. f. Anat. u. Physiol.*, 1905, p. 337.
- SABRAZES et LETESSIER. Procédé de coloration de la névroglie. *Arch. gén. de méd.*, déc. 19, 1905, p. 3220.

## PHYSIOLOGY

- RIGNANO. Sur la transmissibilité des caractères acquis; Hypothèse d'une centro-épigénèse. Alcan, Paris, 1906, 5 fr.
- ROTHMANN. Ueber die Leitung der Sensibilität im Rückenmark. *Berlin. klin. Woch.*, Jan. 8 u. 15, 1906.
- HERING. Grundzüge der Lehre vom Lichtsinn. Engelmann, Leipzig, 1905, M. 1.80.
- A. J. CARLSON. The Physiology of the Cardiac Nerves in the Anthropoids. *Am. Journ. of Phys.*, Jan. 1, 1906, p. 127.
- LANGLEY. Note on the Trophic Centre of the Afferent Fibres accompanying the Sympathetic Nerves. *Journ. Physiol.*, Dec. 30, 1905, p. xvii.
- SUDNIK. Considérations générales sur les réactions de dégénérescence. *Ann. d'Électrobiol.*, Vol. vi., No. 6, 1905, p. 705.
- IOTEYKO. L'analyse mathématique des courbes de fatigue comme procédé de diagnostic dans les maladies nerveuses. *Journ. de Neurol.*, jan. 5, 1906, p. 7.
- SGÖBBO. Manifestations électriques dues au travail musculaire (suite et fin). *Ann. d'Électrobiol.*, Vol. vi., No. 6, 1905, p. 738.
- LANGLEY. On the Reaction of Cells and of Nerve-endings to Certain Poisons, chiefly as regards the Reaction of Striated Muscle to Nicotine and Curari. *Journ. Physiol.*, Dec. 30, 1905, p. 374.
- ROAF and SMITH. A Combined Key and Commutator for Physiological and Psychophysical Purposes. *Journ. Physiol.*, Dec. 30, 1905, p. xiv.

## PSYCHOLOGY

- KASSOWITZ. Nerven und Seele. Perles, Wien, 1905, M. 12.
- LEROY. Le Langage. Essai sur la psychologie normale et pathologique de cette fonction. Alcan, Paris, 1905, 5 fr.
- B. BOSANQUET. Contradiction and Reality. *Mind*, Jan. 1906, p. 1.

- NORMAN SMITH. Avenarius' Philosophy of Pure Experience. *Mind*, Jan. 1906, p. 13.  
 WINCH. Psychology and Philosophy of Play. *Mind*, Jan. 1906, p. 32.  
 H. R. MARSHALL. Presentation and Representation. *Mind*, Jan. 1906, p. 53.  
 C. O. TAYLOR. Über das Verstehen von Worten und Sätzen. *Zeitschrift f. Psych. und Physiol. der Sinnesorgane*, Bd. 40, H. 4, 1905, p. 225.  
 BARDOUX. Essai d'une Psychologie de l'Angleterre contemporaine. Alcan, Paris, 1906, 7 fr. 60.

### PATHOLOGY

- Edinger's Theorie der Entstehung von Nervenkrankheiten durch Funktion. *S. Petersburg. med. Woch.*, Dec. 17, 1905, p. 515.  
 MARINESCO. Recherches sur la régénérescence autogène. *Rev. Neurol.*, déc. 15, 1905, p. 1125.  
 ARTUR SCHÜLLER. Experimentelle Pyramidendurchschneidung beim Hunde und Affen. *Wien. klin. Woch.*, Jan. 18, 1906, p. 57.  
 G. FICHERA. Sulla distruzione dell' Ipofoisi. *Lo Sperimentale*, Jan. 1906, p. 739.  
 A. ALEXANDER. Zur Kenntnis der Rückenmarksveränderungen nach Verschluss der Aorta abdominalis. *Zeit. f. klin. Med.*, 1906, p. 247.  
 VAN GEHUCHTEN. La loi de Waller. *Névrose*, Vol. vii., f. 2, 1905, p. 203.  
 SCHAFFER. Über Fibrillenbilder der progressiven Paralyse. *Neurol. Centralbl.*, Jan. 2, 1906, S. 2.  
 BALLET et LAIGNEL-LAVASTINE. Étude des Lésions cadavériques de l'Écorce Cérébrale de l'homme et du lapin par la méthode de Cajal à l'argent réduit. (Soc. de Neurol.) *Rev. Neurol.*, déc. 30, 1905, p. 1209.  
 DEBUCK et DEROUBAIX. Contribution à l'histopathologie de certaines formes de psychoses appartenant à la démence précoce (Kraepelin). *Névrose*, Vol. vii., f. 2, 1905, p. 161.  
 BRUCE, M'DONALD, and PIRIE. A Second Case of Partial Doubling of the Spinal Cord. *Rev. Neurol. and Psychiat.*, Jan. 1906, p. 6.  
 RABAUD. Pathogénie de la pseudocéphalie et de l'anencéphalie. *Nouv. Icon. de la Salpêtrière*, sept.-oct. 1905, p. 602.

### CLINICAL NEUROLOGY AND PSYCHIATRY

#### GENERAL—

- Bericht ueber die psychiatrische Literatur im Jahre 1904. *Allg. Zeit. f. Psychiat. und psychisch-gerichtliche Medizin*. Literatur Heft zu Band lxii.  
 MITTELHAUSER. Unfall und Nervenkrankung. Marhold, Halle, 1905, M. 1.50.

#### MUSCLES—

- SCHLIPPE. Hochgradige Kontrakturen und Skelattatrophie bei Dystrophie musculorum progressiva. *Deutsche Ztsch. f. Nervenheilk.*, Bd. 30, H. 1-2, 1905, S. 128.  
 KLIPPEL et MAILLARD. Maladie de Recklinghausen avec Dystrophies multiples. (Soc. de Neurol.) *Rev. Neurol.*, déc. 30, 1905, p. 1207.

#### PERIPHERAL NERVES—

- BLISS. Epidemic Multiple Neuritis of Obscure Origin. *Journ. Nerv. and Ment. Dis.*, Dec. 1905, p. 759.  
 LAMY. Polynévrite au cours d'un Cancer Intestinal. (Soc. de Neurol.) *Rev. Neurol.*, déc. 30, 1905, p. 1225.  
 CONZEN. Über Arsenikneuritis. *Neurol. Centralbl.*, Jan. 2, 1906, S. 18.  
 TISSERAND. Luxations du nerf cubital. *Arch. gén. de méd.*, jan. 9, 1906, p. 86.  
 FORBES-ROSS. Two Cases illustrating Sciatica of Abdominal Origin. *Lancet*, Jan. 13, 1906, p. 89.  
 FAUCHON-VILLEPLÉE. Application des courants de H. F. dans les sciaticques névrites. *Bull. de la Soc. française d'Électrothér.*, déc. 1905, p. 225.

## SPINAL CORD—

ORR and ROWS. Lesions of the Spinal Cord, the Result of Absorption from Localised Septic Foci, with a Preliminary Note on an Experimental Research. *Rev. Neurol. and Psychiat.*, Jan. 1906, p. 25.

**Progressive Muscular Atrophy.**—LANNOIS. Atrophie musculaire du type Aran-Duchenne d'origine syphilitique. *Nouv. Icon. de la Salpêtrière*, sept.-oct. 1905, p. 593.

**Tabes.**—E. v. MALAISE. Die Prognose der Tabes Dorsalis. *Monatsschrift f. Psychiat. u. Neurol.*, Bd. 18, p. 233.

G. KÖSTER. Zur klinik und pathologischen Anatomie der Tabes und Taboparalyse des Kindesalters. *Monatsschrift f. Psychiat. u. Neurol.*, Bd. 18, p. 179.

MOBIUS. Neuere Beobachtungen über die Tabes. *Schmidts Jahrbuch*, 1906, H. 1.

DEJERINE et LEENHARDT. Atrophie et Paralysie unilatérale des Muscles du Dos et de l'Abdomen au cours du Tabes. (Soc. de Neurol.) *Rev. Neurol.*, déc. 30, 1905, p. 1218.

CHAUFFARD. Zona chez un ataxique. *Journ. de méd. et de chir.*, nov. 10, 1905, p. 805.

HIRSCHBERG. Über den plötzlichen Tod bei Tabischen. *Neurol. Centralbl.*, Jan. 2, 1906, S. 21.

**Friedreich's Ataxia.**—WHARTON SINKLER. Friedreich's Ataxia, with a report of thirteen cases. *New York Med. Journ.*, Jan. 13, 1906, p. 65.

**Syringomyelia.**—KLIPPEL et MAILLARD. Troubles trophiques des Mains paraissant dus à une Syringomyelie. (Soc. de Neurol.) *Rev. Neurol.*, déc. 30, 1905, p. 1205.

**Disseminated Sclerosis.**—CATOLA. Sclérose en plaques; atrophie cérébelleuse et sclérose pseudo-systématique de la moelle épinière. *Nouv. Icon. de la Salpêtrière*, sept.-oct. 1905, p. 585.

**Cauda Equina.**—BALINT und BENEDICT. Über Erkrankungen des Conus terminalis und der Cauda equina. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 30, H. 1-2, 1905, S. 1.

**Cerebro-Spinal Fluid.**—SARNELE. Beitrag zur Kenntnis der Zytologie der Cerebro-spinalflüssigkeit bei Nervenkranken. *Zeit. f. klin. Med.*, 1906, p. 282.

## BRAIN—

**Meningitis.**—Cerebro-Spinal Meningitis. Leading Article. *Lancet*, Jan. 13, 1906, p. 103.

CHAILLY. Séquelles nerveuses de la méningite cérébro-spinale épidémique. *Thèse*. Maloine, Paris, 1905.

MANDOUL. Un cas de paralysie faciale corticale consécutive à une méningite cérébro-spinale ayant évolué favorablement. *Arch. gén. de méd.*, jan. 16, 1906, p. 152.

B. HEINE. Die Prognose der otogenen Meningitis. *Berlin. klin. Woch.*, Jan. 22, 1906, p. 105.

**Hæmorrhage.**—KARL. Zur Kasuistik der traumatischen Spätopoplexie (Bollinger). *Zeit. f. Heilk.*, 1905, p. 321.

ROUSSY. Hémorrhagie de la Couche Optique. (Soc. de Neurol.) *Rev. Neurol.*, déc. 30, 1905, p. 1227.

**Tumeur.**—ROUSSY. Contribution à l'étude des tumeurs méningées. *Arch. gén. de méd.*, déc. 19, 1905, p. 3211.

GORDINIER and CAREY. A Study of Two Unusual Brain Tumours. *Journ. Nerv. and Ment. Dis.*, Jan. 1906, p. 1.

ROUSSY. Un cas de cholestéatome de la base de l'encéphale. *Rev. Neurol.*, déc. 30, 1905, p. 1183.

HUNZIKER. Beitrag zur Lehre von den intraventriculären Gehirntumoren. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 30, H. 1-2, 1905, S. 77.

**Abcess.**—HEIMANN. Ein Fall von akutem otitischem Schläfenlappenabcess. *Arch. f. Ohrenheilk.*, Bd. 66, p. 303, and Bd. 67, p. 1.

**Syphilis.**—MARCHAND. Du rôle de la syphilis dans les maladies de l'encéphale. *Octave Doin*, Paris, 1906, fr. 3.50.

FALINYI. Über die syphilitische Erkrankung der Basilararterien des Gehirns. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 30, H. 1-2, 1905, S. 44.

- Hemiplegia.**—CLAUDE et LEJONNE. Un cas d'Hémiplégie avec troubles très accusés de la Sensibilité. (Soc. de Neurol.) *Rev. Neurol.*, déc. 30, 1905, p. 1219.
- SOUQUES. Intérêt Clinique et Médico-légal d'un cas d'Hémiplégie Traumatique tardive. (Soc. de Neurol.) *Rev. Neurol.*, déc. 30, 1905, p. 1212.
- BENON. Les troubles Psychiques chez les Hémiplégiques Organiques internés. *Thèse de Paris*, 1905.
- Diplegia.**—STERLING. Zur Kasuistik der Tay-Sachs'schen Krankheit (Idiotismus familiaris amauroticus). *Neurol. Centralbl.*, Jan. 16, 1906, S. 55.
- SPIELMEYER. Über eine besondere Form von familiärer amaurotischer Idiotie. *Neurol. Centralbl.*, Jan. 16, 1906, S. 51.
- General Paralysis.**—INGENIEROS. Classification clinique des syndromes paralytiques généraux. *Rev. Neurol.*, déc. 30, 1905, p. 1173.
- SAILLANT. Contribution à l'étude des Décubitus aigus et chroniques chez les Déments Paralytiques. *Thèse de Paris*, 1905.

#### MENTAL DISEASES—

- KERN. Das Wesen des menschlichen Seelen- und Geisteslebens. Hirschwald, Berlin, 1905, M. 2.40.
- GELPKE. Culturschäden oder die Zunahme der Nerven- u. Geisteskrankheiten. Hygiene des Nervenlebens. Schwabe, Basel, 1905, M. 1.60.
- JOLICEUR. L'Idée de persécution, stigmate de dégénérescence. *Thèse*. Nancy, 1905.
- E. STRANSKY. Dementia tardiva. *Monatsschrift f. Psychiat. u. Neurol.*, Bd. 18, p. 1.
- C. SIBELIUS. Die psychischen Störungen nach akuter Kohlenoxydvergiftung. *Monatsschrift f. Psychiat. u. Neurol.*, Bd. 18, p. 39.
- HEILBRONNER. Über Haftenbleiben und Stereotypie. *Monatsschrift f. Psychiat. u. Neurol.*, Bd. 18, p. 293.
- ISSERLIN. Assoziationsversuche bei einem paresisch begutachteten Fall von epileptischer Geistesstörung. *Monatsschrift f. Psychiat. u. Neurol.*, Bd. 18, p. 419.
- WEYGANDT. Über Idiotie. Carl Marhold, Halle, 1905, M. 2.
- YOSHIKAWA. Ein Fall von Idiotie mit Erweichungsherd in den Zentralganglien des Gehirns. *Monatsschrift f. Psychiat. u. Neurol.*, Bd. 18, p. 282.
- SOKALSKY. Les psychoses aiguës et leur classification. *Ann. méd.-psychol.*, jan.-fév. 1906, p. 5.
- FAUSER. Zur allgemeinen Psychopathologie der Zwangsvorstellungen. *Centralbl. f. Nervenheilk. u. Psychiat.*, Dec. 15, 1905, S. 933.
- BRAUN. Die religiöse Wahnbildung. Mohr, Tübingen, 1906.
- GIMBAL. Les incendiaires (suite). *Ann. méd.-psychol.*, jan.-fév. 1906, p. 39.
- LAURENT. Physionomie et mimique chez les aliénés (suite et fin). *Ann. méd.-psychol.*, jan.-fév. 1906, p. 54.
- MARCUS WYLER. A Précis of the Conditions under which Lunatics are received in Continental Asylums. *Brit. Med. Journ.*, Jan. 13, 1906, p. 78.
- BRIAND. Prophylaxie et traitement de la tuberculose dans les asiles d'aliénés. *Ann. méd.-psychol.*, jan.-fév. 1906, p. 32.
- DUPONT. De la Kleptomanie. *Journ. de Psychol. normale et patholog.*, sept.-oct. 1905, p. 404.
- PARIANI. Il tetano faradico in alcune malattie mentali. *Riv. di Patolog. nerv. e ment.*, Vol. x., f. 11, 1905, p. 497.
- HECHT. A Study of Dementia Præcox. *Journ. Nerv. and Ment. Dis.*, Dec. 1905, p. 763.
- ROCHU. Des Hallucinations dans la Mélancolie et des Phénomènes Hallucinatoires post-mélancoliques. *Thèse de Paris*, 1905.
- KAUFMANN. Schloss Werneck, die Kreis-Irrenanstalt für Unterfranken. Stadel, Würzburg, 1905, M. 2.

#### GENERAL AND FUNCTIONAL DISEASES—

- Epilepsy.**—ONUF. On the Association of Epilepsy with Muscular Conditions Fitting Best in the Cadre of the Myopathies. *Journ. Nerv. and Ment. Dis.*, Jan. 1906, p. 12.
- REYNOLDS. Paramyoclonus Epilepticus. *Rev. Neurol. and Psychiat.*, Jan. 1906, p. 19.
- ROSANHOFF. The Diet in Epilepsy. *Journ. Nerv. and Ment. Dis.*, Dec. 1905, p. 753.

GUY HINSDALE. The value of hydrotherapy in the Treatment of Epilepsy. *Journ. Am. Med. Ass.*, Jan. 20, 1906, p. 175.  
The Epileptic Patient and the Lunacy Laws. Leading Article. *Lancet*, Jan. 20, 1906, p. 169.

**Hysteria.**—A. MATHIEU et J. CH. ROUX. L'hystérie gastrique et ses stigmates périphériques. *Gaz. des Hôp.*, Jan. 11, 1906, p. 39.

PITRES et CRUCHET. Le tic hystérique. *Journ. de Neurol.*, déc. 20, 1905, p. 541.

HÜTTENBACH. Ein Beitrag zur Frage der Kombination organischer Nerven-erkrankungen mit Hysterie. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 30, H. 1-2, 1905, S. 103.

**Traumatic Neuroses.**—SPECHT. Einige Bemerkungen zur Lehre von den traumatischen Neurosen. *Centralbl. f. Nervenheilk. u. Psychiat.*, Jan. 1906, S. 1.

**Reflex Neuroses.**—EGBERT H. GRANDIN. Reflex Neuroses with Special Reference to the Appendix Vermiformis. *New York Med. Journ.*, Jan. 13, 1906, p. 72.

**Paralysis Agitans.**—LAMY. Écriture dans la Maladie de Parkinson. (Soc. de Neurol.) *Rev. Neurol.*, déc. 30, 1905, p. 1226.

**Tic.**—MORTON PRINCE. Case of Multiform Tic including Automatic Speech and Purposive Movements. *Journ. Nerv. and Ment. Dis.*, Jan. 1906, p. 29.

IOTEYKO. Un cas de tics de la face guéri par suggestion. *Journ. de Neurol.*, jan. 5, 1906, p. 1.

**Migraine.**—S. E. JELLIFFE. Aphasia, Hemiparesis, and Hemianesthesia in Migraine. *New York Med. Journ.*, Jan. 6, 1906, p. 33.

**Myasthenia Gravis.**—MICHELL CLARKE. Two Cases of Myasthenia Gravis. *Bristol Med.-Chir. Journ.*, Dec. 1905, p. 308.

**Exophthalmic Goitre.**—J. DRESCHFELD. On Some of the Symptoms and Treatment of Graves' Disease. *Med. Chron.*, Jan. 1906, p. 203.

**Dercum's Disease.**—LE PLAY. Un cas de Maladie de Dercum. (Soc. de Neurol.) *Rev. Neurol.*, déc. 30, 1905, p. 1202.

#### ALCOHOLISM.—

NEILD. A Report on the Tenth International Anti-Alcoholic Congress. *Brit. Journ. Inebriety*, Jan. 1906, p. 127.

PERAZZOLO. Il crepitio delle falangi negli alcoolisti (segno di Quinquaud). *Riv. di Patolog. nerv. e ment.*, Vol. x., f. 11, 1905, p. 524.

#### SPECIAL SENSES AND CRANIAL NERVES—

HÜBNER. Ueber die psychische und sensible Reaktion der Pupillen. *Centralbl. f. Nervenheilk. u. Psychiat.*, Dec. 15, 1905, S. 945.

BABINSKI. De l'influence de l'Obscurité sur le Réflex des Pupilles à la lumière et de la pseudo-abolition de ce réflexe. (Soc. de Neurol.) *Rev. Neurol.*, déc. 30, 1905, p. 1214.

ANDERSON. The Paralysis of Involuntary Muscle. Part III. On the Action of Pilocarpine, Physostigmine, and Atropine upon the Paralyzed Iris. *Journ. Physiol.*, Dec. 30, 1905, p. 414.

STRANSKY. Zur Kenntnis des assoziierten Nystagmus. *Neurol. Centralbl.*, Jan. 2, 1906, S. 15.

SCHLESINGER. Ein Fall von doppelseitiger, umschriebener Gesichtsatrophie. *Archiv f. Kinderheilk.*, Bd. 42, 1906, p. 374.

GOWERS. The Influence of Facial Hemiatrophy on the Facial and other Nerves. *Rev. Neurol. and Psychiat.*, Jan. 1906, p. 1.

SCHLESINGER. Ein nicht beschriebenes Symptom der Gaumenlähmung (Änderung der Sprachstörung im Liegen und in aufrechter Körperhaltung). *Neurol. Centralbl.*, Jan. 16, 1906, S. 50.

#### MISCELLANEOUS SYMPTOMS—

VALOBRA. Difformité congénitale des membres. *Nouv. Icon. de la Salpêtrière*, sept.-oct. 1905, p. 560.

PÁRHON, SHÜNDA, et ZALPLACHTA. Sur deux cas d'achondroplasie. *Nouv. Icon. de la Salpêtrière*, sept.-oct. 1905, p. 539.



- PORAK et DURANTE. Les micromélie congénitales. Achondroplasie vraie et dystrophie périostale. *Nouv. Icon. de la Salpêtrière*, sept.-oct. 1905, p. 481.
- ÉTIENNE. Arthropathies nerveuses et rhumatisme chronique. *Rev. Neurol.*, déc. 15, 1905, p. 1137.
- ÉTIENNE. Rôle du Froid Intense dans la Pathogénie des Acropathies. *Arch. gén. de méd.*, déc. 26, 1905, p. 3265.
- BRISAUD ET MOUTIER. Œdème Éléphantiasique des membres inférieurs, Astéréognosie, Surdité, origine centrale de ces troubles. (Soc. de Neurol.) *Rev. Neurol.*, déc. 30, 1905, p. 1204.
- PELLETIER et MARIE. Les Membres fantômes chez les amputés délirants. Colin, Paris, 1905.
- DUFOUR. Du syndrome "deviation conjuguée de la tête et des yeux" (théorie sensorielle). (Soc. de Neurol.) *Rev. Neurol.*, déc. 30, 1905, p. 1199.
- BYROM BRAMWELL. A Series of Lectures on Aphasia. Lecture I. *Lancet*, Jan. 13, 1906, p. 71.
- LOEWY. Mikrographie durch hemiplegischen Anfall wahrscheinlich in folge auf die Schreibkoordination beschränkter Rigidität. *Monatsschrift f. Psychiat. u. Neurol.*, Bd. 18, p. 372.

#### TREATMENT\*—

- ROSS. On the Relief of certain Headaches by the Administration of one of the Salts of Calcium. *Lancet*, Jan. 20, 1906, p. 143.
- SCHLOESSER. Zur Behandlung der Neuralgien durch Alkoholeinspritzungen. *Berlin. klin. Woch.*, Jan. 15, 1906, p. 82.
- BERILLON. Des anesthésiques et en particulier de la scopolamine envisagés comme adjuvants à la suggestion hypnotique. *Journ. de Neurol.*, jan. 5, 1906, p. 13.
- CONTET. Gymnastique médicale et Rééducation. *Arch. gén. de méd.*, déc. 26, 1905, p. 3283.
- LOUYRIAC. De la sympathicectomie dans les névralgies faciales. *Thèse*. Storck et Cie, Lyon, 1905.
- RICHARD LAKE. A Case of Operation on the Vestibule for the Relief of Vertigo; together with a description of the Flap employed in order to obtain a better view of the parts during operation. *Lancet*, Jan. 6, 1906, p. 26.
- ADOLF LORENZ. Der Indikationen zur Sehnenverpflanzung. *Wien. med. Woch.*, Jan. 13, 1906, p. 118.
- P. L. BRETON. Treatment of the Results of Infantile Paralysis. *Journ. Am. Med. Ass.*, Jan. 6, 1906, p. 26.

\* A number of references to papers on Treatment are included in the Bibliography under the individual Diseases.

# **Review**

of

# **Neurology and Psychiatry**

---

## **Original Articles**

### **THE PATHOLOGY OF GENERAL PARALYSIS OF THE INSANE.**

By **W. FORD ROBERTSON, M.D.,**

Pathologist to the Scottish Asylums.

(**The Morison Lectures for 1906.**)

#### **LECTURE II.**

Delivered on 26th January 1906.

TO-DAY I wish to deal chiefly with the results of an experimental enquiry into the action of the living blood and of the blood-serum upon pure cultures of diphtheroid bacilli isolated from cases of general paralysis. As explained at the end of the previous lecture, Dr M'Rae and I were led to take up this study because we had reason to suspect that certain indistinct granular or rod-like bodies observed in the cerebro-spinal fluid, in the walls of the cerebral vessels, in the blood and in the urine, were really diphtheroid bacilli that had suffered from the effects of a lysogenic or solvent action. We have also, however, had in view the possibility of being able to discover some specific reaction on the part of the blood or blood-serum of the general paralytic towards the diphtheroid bacillus, for, if such a specific reaction were found, it would not only serve to establish our hypothesis regarding the etiology of general paralysis, but it

would at the same time probably furnish a method of serum diagnosis.

Before entering upon this subject I must clear the way by describing the morphological and biological characters of the bacillus with which we have worked. When, in our published communications, my colleagues and I have spoken of a diphtheroid bacillus, we have simply meant a bacillus that has the general cultural and morphological features and the staining reaction to Neisser's method which characterise the Klebs-Löffler bacillus. The organism that is so abundant in cases of general paralysis is certainly neither Hoffmann's bacillus, nor the xerosis bacillus. It forms acid when grown in glucose broth, and it also differs from these two species in its morphological characters. Now it is laid down by such authorities as Muir and Ritchie that an organism differing from the diphtheria bacillus solely in its want of virulence must be regarded merely as a diphtheria bacillus in an attenuated condition, and should be spoken of as such. Neisser and several other authorities have expressed a similar opinion. We have therefore, I think, been perfectly justified in provisionally regarding the organism we have studied as an attenuated form of the Klebs-Löffler bacillus. In doing so we have left it an open question if the organism differs in certain essential respects yet to be discovered from the attenuated form of the Klebs-Löffler bacillus, and is therefore a special bacillus. Our more recent observations strongly incline us to the view that it is a special organism; but, whether this supposition should turn out to be correct or not, if an organism of this nature is really the essential pathogenic agent in general paralysis and tabes dorsalis, as we believe we have evidence to prove, then surely it is deserving of a special name. We therefore propose to refer to it as the *bacillus paralyticans*.

We have studied very numerous cultures of this bacillus, and, in a large number of instances at least, we had good grounds for believing that we were dealing with an organism that had been exerting a pathogenic action upon the patient from whom it was isolated. As has been indicated, it has the general morphological characters of the Klebs-Löffler bacillus. It is capable of assuming the granular, barred, and solid-colour forms of Wesbrook. Perhaps its most striking characteristic is

its polymorphism, and in this respect it would appear even to excel at least the virulent form of the Klebs-Löffler bacillus. When grown upon bynohæmoglobin agar at 37° C., it constantly shows more or less distinct metachromatic granules in preparations stained by Neisser's method. When grown upon blood-films it has the same appearance. Cultures upon the ordinary agar medium very rarely show any metachromatic granules. Individual strains of the organism differ greatly in regard to the size of the metachromatic granules they are capable of displaying. Some have very large granules which, after two days' growth of the organism upon bynohæmoglobin agar at the ordinary temperature, present in Neisser preparations a peculiar metallic lustre which we have not been able to observe in bacilli isolated from cases of acute diphtheria. The organism grows feebly under anærobic conditions, but, nevertheless, it is capable of multiplying rapidly with a very limited supply of oxygen. The appearance of the *bacillus paralyticans* varies not only with the medium upon which it is grown, but also with the temperature employed. Strains of the organism which, when grown at a temperature of 37° C. upon bynohæmoglobin agar, show prominent metachromatic granules, when cultivated at a temperature of 30° exhibit extremely minute granules. This difference is accentuated if the cultures are made upon blood-films. At the lower temperature the bacilli may then be entirely devoid of metachromatic granules.

In old cultures, clubbed and elongated forms are very abundant. Short threads may often be observed. It is hardly open to question that the filamentous organism, already described as having been observed in the tissues, is a special form of this bacillus. It has been found invading the tissues in four rats fed, or injected, with cultures of the bacillus, and in one of these animals the segments of the threads show metachromatic granules exactly like those of the bacillary form. As a rule, however, metachromatic granules are not visible. We have endeavoured to determine the precise conditions under which the thread form is assumed. There are grounds for believing that this special morphological character is, in part at least, one that is gradually impressed upon the organism by environmental influences, for, in several instances, the bacillus has shown a strong tendency to form short threads during the first two or three days

after having been isolated from the patient, irrespective of the culture medium employed and at the normal temperature. We have ascertained that threading is not due to the presence of the toxic products of the bacillus, to growth at a low temperature, or to limitation of the supply of oxygen. It appears to be in large part due to an abnormally high temperature, but also to be favoured by semi-anærobic conditions. When the bacilli are grown upon a blood-film at a temperature of 42° C. in a tube sealed with hard paraffin, they become attenuated and elongated, and tend to cohere by their extremities so as to form at first chains and then distinct filaments. The threading is well marked in two or three days, but it becomes still more distinct if cultivation is continued upon a fresh blood-film under the same conditions. The segments forming the threads generally show two or three minute metachromatic granules.

In view of the extreme variability of the morphological characters of this bacillus, it is not to be expected that it should always appear in the general paralytic in the form with metachromatic granules. It has indeed been definitely ascertained that in the living body it is the exception for the bacillus to present these granules. Even when growing in catarrhal secretions it shows them only occasionally. When it invades the tissues it is almost constantly devoid of visible metachromatic granules. It then generally assumes a simple granular and often a diplo-bacillary form. This corresponds to the appearance that the organism tends to take when grown upon the ordinary agar medium at the normal temperature, or upon a blood-film at a subnormal temperature. In other instances the invading bacillus, as already indicated, assumes a thread form, and this special morphological change is to be attributed in part at least to the occurrence of pyrexia.

By way of further preface it is necessary that I should say a word regarding the mechanism of natural and acquired immunity. Here, as in other departments of progressive science, there is much conflict of opinion. The facts that bear upon the observations I am about to record are, however, few in number and among those that are pretty generally admitted. When foreign invaders, such as bacteria, pass through the first line of defence formed by the skin and mucous membranes, they are normally engulfed by leucocytes and destroyed by the solvent action of

certain intra-cellular ferments. Some authorities also attach importance to the extra-cellular action of similar ferments (that is to say, to the alexines or complements), and of certain substances termed inter-bodies which combine with the invading organisms. Such in brief is the mechanism of natural immunity. In acquired immunity, that is to say the establishment of an increased power of resistance to a particular micro-organism or to some special toxine, the mechanism is more complicated. In response to the inimical stimulus the cells of the body, after a certain interval, produce specific anti-bodies capable of neutralising the toxins or of combining with the micro-organisms, which are thereby so affected that they are quickly dissolved by the alexines. Metchnikoff, in opposition to many other authorities, holds that this process is, in natural conditions, entirely an intra-cellular one, although when the blood is shed and undergoes coagulation the protective substances soon pass into the blood-serum. I specially mention this view because certain of the results we have obtained with the *bacillus paralyticus* harmonise with it. Recently, E. A. Wright has shown that the power of the leucocytes to take up bacteria is dependent upon the existence of certain substances in the blood plasma or blood serum, which he has termed "opsonins."

One of the points which we have specially studied is the phagocytic action of the leucocytes upon the *bacillus paralyticus*. The method we have employed is as follows:—

*Apparatus, etc., required.*—Specimen tubes of 2.5 c.c. capacity (with corks), carefully cleaned and sterilised by dry heat. Portable incubator for maintaining tubes at temperature of 37° C. Large platinum loop (ring), 4 mm. in diameter (No. 24 wire). Small platinum loop, 1 mm. in diameter. Bacillary emulsion prepared by mixing one small loopful of 24-hour culture (upon bynohæmoglobin agar) of *bacillus paralyticus* (i.e. a diphtheroid bacillus isolated from the blood or cerebro-spinal fluid of a patient suffering from general paralysis) with 5 c.c. of .75 per cent. salt solution in distilled water. Sterilised vaseline.

Wash and dry dorsum of thumb. Apply absolute alcohol and, after a minute or so, allow it to evaporate. To the dry surface apply a drop of hot sterilised vaseline. Allow the vaseline to solidify and then prick the thumb through the vaseline with sterilised needle. Place four large loopfuls of blood in specimen tube, and immediately reinsert cork. Maintain the tube at temperature of 37° C. After half an hour, remove the clot with the aid of platinum loop. To the mixture of blood-serum and corpuscles remaining in the tube, add one large loopful of bacillary emulsion (heated to 37° C.) and mix. Incubate for time desired (see below), and then, after having stirred the contents of the tube, make coverglass films of the fluid with the aid of the small platinum loop. Allow the films, which must be very thin, to dry

in air, and then fix them for two minutes with absolute alcohol. Stain with carbol thionin, Löffler's methylene blue, or other suitable staining reagent.

Two separate actions require to be studied, namely, the power of the polymorpho-nuclear leucocytes to take up the bacilli, and the power of these leucocytes to dissolve the bacilli when engulfed.

The first action was studied in films prepared after incubation at 37° C. for thirty minutes (Fig. 1), the second after incubation for three hours. We have found that the power to take up the bacilli is extremely variable, both in control blood and in the general paralytic's blood. No constant alteration in this power could be recognised in the latter.

On the other hand, the power to dissolve the bacilli taken up has, in most instances, been distinctly greater on the part of the leucocytes of the general paralytic than on that of the leucocytes of the controls. Bacilli which have not been taken up by leucocytes remain, with few exceptions, perfectly normal in appearance after three hours' incubation. The intra-cellular changes are, however, within this time always very considerable (Fig. 2). It would appear that, as maintained by Metchnikoff to be the general rule, the bacteriolytic action is in this case essentially an intra-corpuseular one. The dissolving organisms show a progressive diminution in their affinity for the ordinary staining reagents (Fig. 3). In the last distinguishable phase of this bacteriolytic process the organisms appear as faintly tinted rods, and are generally somewhat attenuated. It is only recently that we have succeeded in sufficiently perfecting the method of demonstrating this bacteriolytic action to warrant the making of comparative observations with it in a series of cases, and therefore the number of these observations that we can as yet record is somewhat small. In films fixed with absolute alcohol and stained for ten minutes with Löffler's methylene blue, we have estimated the percentage of altered bacilli among those engulfed by the leucocytes. This percentage we term the *intra-corpuseular bacteriolytic index*. The following table shows the results obtained in twelve cases of general paralysis and in six control cases :—

## INTRA-CORPUSCULAR BACTERIOLYTIC INDICES.

<i>I. General Paralysis.</i>			<i>II. Controls.</i>		
1. A. (w.).	.	77	1. V. (w.) Adolescent In-		
2. R. (w.).	.	64	sanity	.	16
3. R. (w.).	.	67	2. I. (w.) Imbecility	.	31·5
4. K. (w.).	.	63	3. Nurse J.	.	5·8
5. C. (w.).	.	78	4. Nurse H.	.	15
6. W. (w.).	.	70·6	5. S. (w.) Adolescent In-		
7. S. (m.).	.	86	sanity	.	10·5
8. W. (m.).	.	64·4	6. M. (w.) Adolescent		
9. A. (m.).	.	43·5	Insanity	.	13
10. D. (m.).	.	80	<i>III. Clinical Diagnosis Doubtful.</i>		
11. G. (m.).	.	65	1. W. (w.).	.	15
12. N. (m.).	.	51			

We have also studied the bacteriolytic or lysogenic action of the blood serum upon the *bacillus paralyticans*. After the blood has been allowed to stand for twenty-four hours, the bacteriolytic substances are contained in the serum owing to the disintegration of the white corpuscles. The elaboration of a suitable technic by means of which to compare the bacteriolytic power in different cases has proved somewhat difficult. It has, however, already been possible to determine in several cases of general paralysis that the solvent action of the blood serum, like that of the active leucocytes, was greater than the solvent action of the serum of a normal person.

After having thus studied experimentally the phagocytic and bacteriolytic actions of the blood upon the *bacillus paralyticans*, Dr M'Rae and I endeavoured to ascertain if there was any evidence of the occurrence of similar processes in the tissues and body fluids of general paralytics. Every case studied with this object has given the same answer, an emphatic "Yes."

Diphtheroid bacilli, more or less altered by lysogenic action, are present in great numbers in the catarrhal pneumonic foci that occur in most general paralytics who die in congestive attacks (Fig. 4). They are contained chiefly, but not exclusively, in the leucocytes that fill the alveoli. They have also been found in the blood stream in the neighbourhood of such pneumonic foci. They are also generally demonstrable, sometimes in great numbers, in the adventitial spaces of the inflamed cerebral



vessels and in meshes of the pia-arachnoid. Further, they can be observed in films made from the blood of the living paralytic, more especially when the patient is in a congestive attack (Figs. 7). They can also be detected in the centrifuge deposit from the cerebro-spinal fluid. They may be present in great numbers in the urine of paralytics, especially during congestive attacks. If 5 c.c. of the fresh urine are centrifuged, and if films of the deposit are then stained by Neisser's method or with carbol thionin, abundant organisms are generally to be observed. These may be of various kinds, but in several cases we have found that to all appearance only dissolving diphtheroid bacilli were present. That this was really so was demonstrated in a striking way in one of our cases in which these altered bacilli were abundant in the centrifuge deposit (Fig. 6). We made cultures from the deposit, and after 48 hours' incubation there was on first inspection seemingly no growth of any kind. On closer examination, however, a single minute colony was observed. It proved to be a pure growth of a diphtheroid bacillus. In cases of simple general paralysis it is unusual to find among the diphtheroid bacilli in the centrifuge deposit from the urine any that show metachromatic granules. In cases of general paralysis combined with *tabes dorsalis* it is, however, different. In all of seven such cases that we have been able to examine at the Royal Edinburgh Asylum, Dr M'Rae and I have found abundant living bacilli with distinct metachromatic granules also to be present. I shall discuss the significance of these facts in the concluding lecture.

The experimental study of the action of the living blood upon the *bacillus paralyticans* has, however, led to another result of a different kind. It has taught us how to grow the organism from the blood and cerebro-spinal fluid of the living general paralytic. More than four years ago Dr M'Rae, Dr Jeffrey, and I began to make attempts to grow the bacillus from the blood, for we felt that it should be possible to do so if the hypothesis we were testing was correct. Every method that we could think of has been tried from time to time during these years, but, until quite recently, all in vain. It is now clear, in the light of our experimental observations, that we failed to get cultures chiefly for the reason that we incubated the tubes with

as little delay as possible. By so doing we were really adopting the most certain method of completing the destruction of the few living bacilli that might be present. From several cases of general paralysis we have recently placed fresh blood upon byno-hæmoglobin agar and allowed the tubes to remain in the cold for twenty-four hours before incubating them at 37° C. By thus delaying the incubation, two important ends are attained. First, the leucocytes are killed and their phagocytic action therefore abolished; and second, time is allowed for the alexines, which, with the disintegration of the leucocytes, have passed into the blood serum, to be destroyed by contact with the dead organic matter contained in the culture medium. When the tubes are placed in the incubator the blood has more or less completely lost its bactericidal properties, and any living organisms it may contain are able to multiply.

By using this method, or slight modifications of it, we have succeeded in obtaining pure cultures of a diphtheroid bacillus from the blood in four cases of general paralysis. The first case was that of a woman in a congestive attack from which she recovered. The growth of the bacillus was at first very feeble, but in sub-cultures it has gradually increased in vigour (Fig. 5). The second case was that of a woman, also in a congestive attack, which proved fatal a few days later. In this case only extremely feeble colonies were obtained. The bacillus was readily recognised in film preparations, but it was found impossible to obtain a growth in sub-cultures. The third case was that of a man suffering from general paralysis combined with tabes dorsalis. The disease was progressing rapidly, but the patient was not in a congestive attack. In this case also the growth obtained was extremely feeble, but we still have the bacillus growing in sub-cultures. The fourth case was one in which the patient had been a tabetic for some years before he developed the signs of general paralysis. He was suffering from a congestive attack which proved fatal three days later. In this case also the growths were very feeble, but sub-cultures have been successful. We have on several occasions observed this initial feebleness of growth in cultures of diphtheroid bacilli isolated post-mortem from internal organs. There is reason to believe that it is an effect of the previous bacteriolytic action of the patient's blood. In such instances the bacillus, as a rule,

increases in vigour in successive sub-cultures, but sometimes it refuses to continue to grow. We have ascertained that a feeble diphtheroid bacillus can be invigorated by being sub-cultured upon blood-films.

Dr M'Rae and I have made cultures from the centrifuge deposit from the cerebro-spinal fluid withdrawn by lumbar puncture in four cases of general paralysis with negative results, but in each of these instances the tubes were incubated at once, and the patients were not suffering from congestive attacks. Quite recently, two patients suffering from congestive attacks have been examined in the same way, excepting that the tubes, after having been inoculated with the deposit, were allowed to remain cold for several hours. From both we have obtained pure growths of a diphtheroid bacillus. In the first case the colonies were numerous and the growth fairly vigorous. In the other the growth was extremely feeble.

I come next to the observations that my colleague and I have made with the object of obtaining, if possible, evidence of some specific action of the blood of the general paralytic upon the *bacillus paralyticans*. I am not quite sure that we can yet say that we have succeeded, but we have certainly come very near doing so.

We have tried agglutination tests, using chiefly the technic devised by Dr M. H. Gordon, and employed by him with some success in an experimental research with the *bacillus diphtheriae*. We have, however, failed to obtain results of a distinctive nature. In our experience the bacilli form clumps so readily, even in normal blood-serum, that it is difficult to be certain of the occurrence of any specific agglutinative action in the serum of the general paralytic.

We have also endeavoured to compare the bacteriolytic power of the blood-serum of the general paralytic with that of control cases. As already indicated, we have found it extremely difficult to devise a serviceable technic, and all I can say at present is that we have in several instances found the power of the twenty-four hour serum of the general paralytic to dissolve the *bacillus paralyticans* to be distinctly greater than that of the serum of a normal individual.

We have had more definite success in estimating the intracorpuseular bacteriolytic power. The method employed has

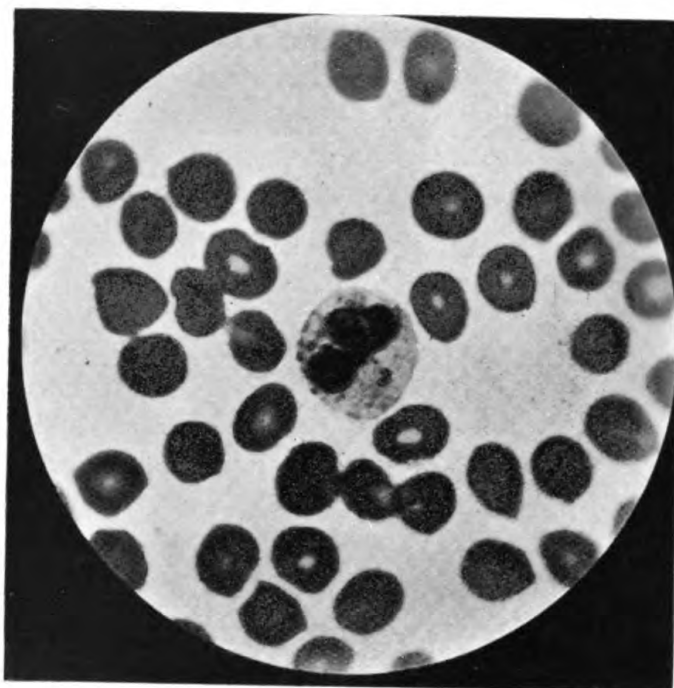


FIG. 1.

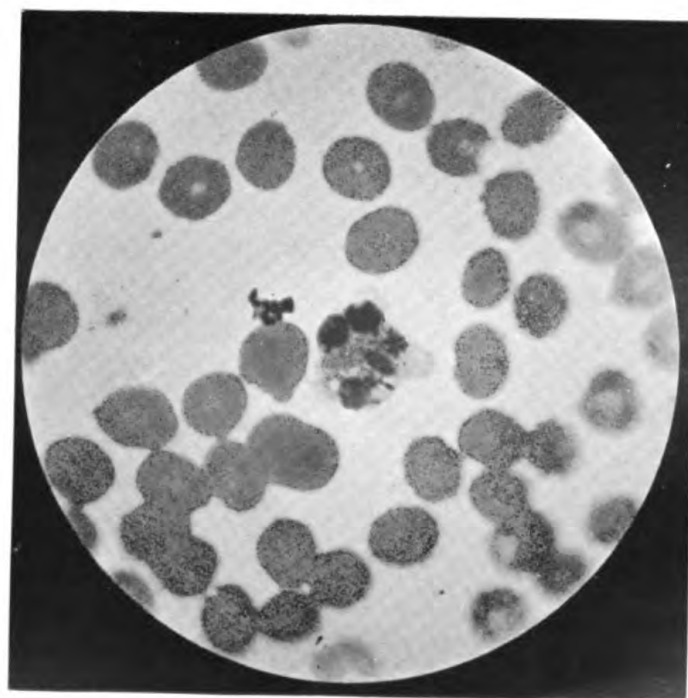


FIG. 2.





FIG. 3.

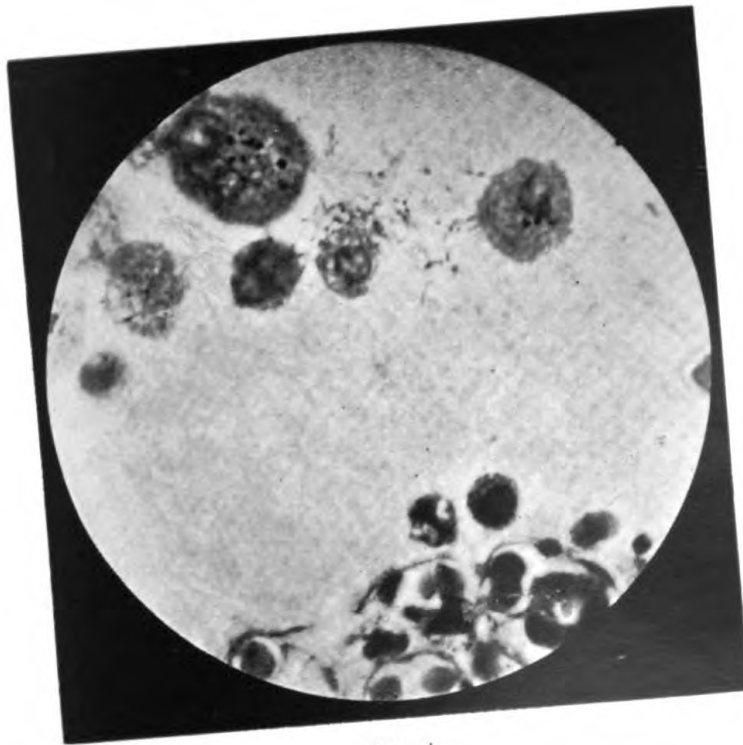


FIG. 4.



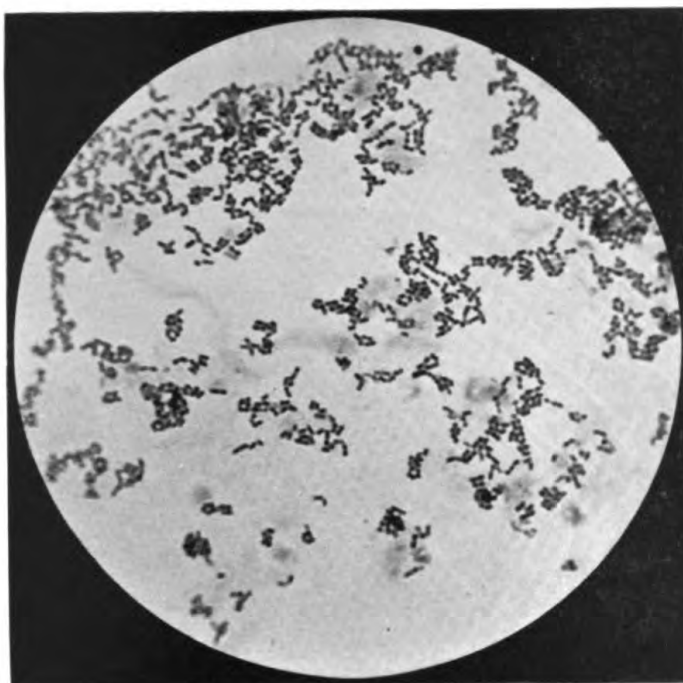


FIG. 5.

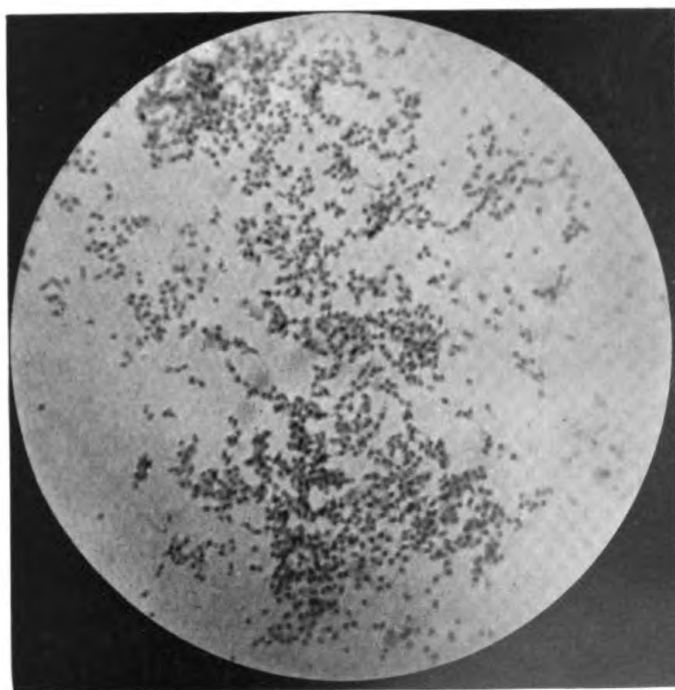


FIG. 6.







FIG. 7.



already been described, and the results obtained have been given in tabular form. If these initial results should be confirmed in a sufficiently extensive series of cases, the reaction would form the basis of a method of serum diagnosis of general paralysis.

The results of the researches of Wright, Bulloch, and others upon the "opsonic" action of the blood-serum, suggested to us that it might be worth while to apply some similar test in this investigation. We have not, however, used Wright's technic for the reason that we had already in use our method of studying the phagocytic action of the leucocytes, and it readily adapted itself to the purpose we had in view, namely, to ascertain if the addition of a definite proportion of the blood-serum of a general paralytic to normal blood-serum containing active leucocytes would increase the power of these corpuscles to take up the *bacillus paralyticans*. The blood, the serum of which was to be tested, was obtained in a sterilised glass tube with capillary ends. The ends were then sealed by means of heat. After twenty-four hours the contents of the tube were centrifuged, and the clear cell-free serum was pipetted off and placed in a sterilised specimen tube (2.5 c.c. capacity). This tube was then corked and placed in the incubator. Two tubes of normal blood were obtained in the way already described, and kept at a temperature of 37° C. After half an hour the clots were removed. To one tube, forming the control, there was added one large loopful of bacillary emulsion (heated to 37° C.); to the other there were added a similar loopful of bacillary emulsion and one loopful of the serum to be tested. Both tubes were incubated for 40 minutes. Films were then made from each and stained with carbol thionin or with Löffler's methylene blue. The control and experimental films were compared with regard to (1) the percentage of polymorpho-nuclear leucocytes containing bacilli, and (2) the average number of bacilli in each leucocyte.

In a test of this kind there is inevitably a considerable margin of error, for at least two reasons. First, the result may be affected by slight differences in the quantity of the bacillary emulsion added respectively to the control tube and to the tube containing the serum to be tested; and, second, the result may be affected by slight differences in temperature, as it has been proved that the power to take up the bacilli is very greatly lessened by lowering of the temperature even 3 or 4 degrees below

the normal. Every effort has, of course, been made to render the conditions uniform.

## TEST IV.

*Difference between Experiment and Control.*

				Percentage of Polymorphs containing Bacilli.	Aver. Number of Bacilli in each Leucocyte
I. GENERAL PARALYSIS—					
1. S. (m.)	.	.	.	+ 38	+ 2·2
2. L. (m.)	.	.	.	+ 56	+ 3·6
3. A. (m.)	.	.	.	+ 12	+ ·9
4. D. (m.)	.	.	.	+ 9·9	+ 2·8
5. G. (m.)	.	.	.	- 5	+ ·5
6. W. (w.)	.	.	.	+ 5	+ ·5
7. C. (w.)	.	.	.	+ 40	+ 4·9
8. K. (w.)	.	.	.	- 2	+ ·4
9. A. (w.)	.	.	.	+ 13·5	+ 1·5
10. R. (w.)	.	.	.	+ 9	+ 3·5
II. CONTROLS—					
1. V. (w.) Adolescent Insanity	--	14·8	.	.	- 2·9
2. K. (w.) „ „	+	33·2	.	.	+ 2·6
3. F. (w.) Epilepsy .	+	6	.	.	+ 1·6
4. H. (w.) Adolescent Insanity	-	10	.	.	+ ·3
5. Nurse A.	.	+	7	.	+ 1
6. Nurse S.	.	+	1	.	- ·2
7. Nurse M.	.	-	7	.	- ·7

The results obtained are perhaps those that might have been predicted on the ground of what is already known. Dr Wright, from his studies upon the subject of opsonic action, has come to the following conclusion :—“ In one class of infections the opsonic power with respect to the infecting micro-organisms hardly varies from day to day, remaining always inferior to that of the normal blood. In another class of infections, the opsonic power is continually fluctuating, the range of variation being very far below the normal and very far above the normal. These categories of infections correspond respectively to strictly localised and systemic infections.”<sup>1</sup> As in general paralysis, there is not merely a local

<sup>1</sup> The *Lancet*, December 2, 1905.

infection, but a succession of systemic invasions, the disease would come under the second class, and we should expect to find considerable variation in the opsonic power of the blood in relation to the infecting organism. It may be that very high readings are indicative of stimulation of certain specific resisting mechanisms, but this conclusion is hardly warranted by the results as yet obtained.

Whilst a trustworthy method of serum diagnosis would unquestionably be very useful in dealing with suspected cases of general paralysis, it seems not improbable that more direct methods of bacteriological diagnosis may become generally available. They may already be said to be available in some cases. There can be little room for doubt that the patient is suffering from the paralytic toxæmia if diphtheroid bacilli can be grown from his blood or from his cerebro-spinal fluid, or if they can be detected in microscopical preparations of the blood or of the centrifuge deposit from the cerebro-spinal fluid. The presence in the urine of great numbers of diphtheroid bacilli that have suffered lysogenic action must, I think, also be regarded as a positive sign. Lastly, if, in addition to altered bacilli, the centrifuge deposit from the urine shows very abundant diphtheroid bacilli with metachromatic granules, then the case is one of *tabes dorsalis*, or at least will soon manifest the recognised signs of this disease.

There are still some additional facts that I wish to record, but they will be more conveniently dealt with when I endeavour, on the ground of the results of our investigations, to formulate new and definite conclusions regarding the etiology and pathogenesis of general paralysis and *tabes dorsalis* in the next lecture.

#### DESCRIPTION OF FIGURES.

FIG. 1.—Polymorpho-nuclear leucocyte containing two unaltered diphtheroid bacilli. Experimental observation; normal blood corpuscles and emulsion of diphtheroid bacilli; incubation for half an hour. Carbol thionin.  $\times 1000$ .

FIG. 2.—Polymorpho-nuclear leucocyte containing numerous diphtheroid bacilli, most of which are altered by lysogenic action. Group of unaltered bacilli lying free. Experimental observation; normal blood corpuscles and emulsion of diphtheroid bacilli; three hours' incubation. Löffler's methylene blue.  $\times 1000$ .

FIG. 3.—Drawing of diphtheroid bacilli in films made from mixture of normal blood corpuscles and emulsion of diphtheroid bacilli after three hours' incubation. To show the changes produced in the appearance of the bacilli by lysogenic action. Above there are shown examples of unaltered bacilli, below examples of altered bacilli to be observed within the protoplasm of many of the leucocytes. The organisms to the left are from a preparation stained by Neisser's method, those to the right from one stained with carbol thionin.

FIG. 4.—Group of altered diphtheroid bacilli in alveolus of lung of general paralytic who died in a congestive attack. Carbol thionin.  $\times 1000$ .

FIG. 5.—Diphtheroid bacillus isolated in pure culture from the blood of a general paralytic suffering from a congestive attack which did not prove fatal. Two days' growth upon blood-film. Carbol thionin.  $\times 1000$ .

FIG. 6.—Centrifuge deposit from urine of general paralytic in third stage. Neisser's method. Shows diphtheroid bacilli considerably altered by lysogenic action.  $\times 1000$ .

FIG. 7.—Leucocyte in blood of general paralytic in a congestive attack. Löffler's methylene blue.  $\times 1000$ . Shows in the protoplasm a body which under the microscope can be recognised to be a diphtheroid bacillus that stains faintly. A pure growth of a diphtheroid bacillus was obtained from the blood of this patient.

---

## A CASE OF CHRONIC PROGRESSIVE DOUBLE HEMIPLEGIA.

By E. FARQUHAR BUZZARD, M.D., M.R.C.P.,

Assistant-Physician (late Pathologist) to the National Hospital  
for the Paralysed and Epileptic ;

and

STANLEY BARNES, M.D., M.R.C.P.,

Pathologist to the Queen's Hospital, Birmingham ; late House Physician  
to the National Hospital for the Paralysed and Epileptic.

CASES of this condition in an early stage are so comparatively rare, that it appeared worth while to record an account of one which recently died in the National Hospital for the Paralysed and Epileptic. We wish to express our indebtedness to Dr

Hughlings Jackson for his kindness in allowing us to make use of this case.

Mary C., aged 53, was admitted to the National Hospital, Queen Square, London, on July 25, 1902, complaining of "hysteria," laughing at nothing, and loss of power in walking.

Her family history was quite good, there having been no cases of insanity, fits, or other nervous disease. Her previous health had always been good before the onset of the present illness. She had never had crying fits or fainting in childhood, and although she said that she had never taken regular exercise, she had enjoyed good health. Menstruation began at the age of 15, and had continued to the age of 43.

The present illness came on gradually after a fall, 10 years ago, at the age of 43. The patient was playing a game in the house when she fell down in a sitting position. She felt some pain in the back for a few days, but when this had worn off she appeared to have quite recovered. From that date forward however, she seemed to gradually begin to lapse into her present condition. The attacks of uncontrollable laughter were the first symptoms to appear, and they have gradually become more and more marked with each successive year. At such times, she says, she does not think of laughable things, nor does she know at what she laughs: she is conscious all the time of the absurdity of thus laughing and feels a "fool" to do so, but she has not sufficient control to prevent herself from so doing. In these attacks she does not feel at all happy, "as it makes her feel so stupid."

She noticed weakness of the legs beginning about two years ago, and this has gradually progressed up to the date of admission to hospital. She can still get about without assistance, but not so quickly as before, and she tires after a very short walk.

Her speech, too, has altered in character. From her description, it is evident that in this respect she suffers partly from loss of ideation and partly from difficulty in pronouncing her words: like the other symptoms, this has become progressively worse since it was first noticed.

There has never been any headache or vomiting, nor any trouble with her eyesight. The sphincters have acted normally throughout.



On admission, she was a rather short, stout woman, with dark hair streaked with grey, and looking a little older than her years.

*Facial Aspect.*—As she lay in bed, unaware that she was being observed, the face was symmetrical and was only noticeable for some loss of the natural expression. When addressed, the face lapsed into a much wrinkled and rather stupid-looking smile, in which the muscles about the angles of the mouth were fully contracted without a similar contraction in those of the upper part of the face, so that no change occurred in the width of the palpebral fissures. There was thus no play of features around the eyes, and the smile reminded one rather of that of an imbecile than of that of an amused adult. At times, on merely asking her an ordinary question—*e.g.* “How are you to-day?”—the smile would broaden out to a grin from ear to ear, and she would give a “He, haw,” and at once bury her head in the pillows and shake with laughter. After repeated observations, it was clear that the laugh was never a quick response to any question, not so quick as would have occurred in a normal woman, even when she was obviously prepared to laugh before she was addressed: the stages of the laugh could always be followed as they gradually rose to a maximum. When asked to raise her eyebrows, or perform any other facial movement, she could do so symmetrically and fully, but the action never had the normally quick response of the healthy individual. All the facial movements seemed to be awkward and slow.

*Attitude on Standing and Walking.*—In standing, the back was always a little bowed, and the head thrown forwards, the chin rather protruding. The arms were at the sides with the elbows a little flexed and the hands in a state of general partial flexion. In walking, she shuffled along with small steps, the feet being dragged along and sometimes scraping the floor. The knees were bent in walking, but not so much as usual. The whole gait had an aspect of stiffness, and although there was no tremor or propulsion, yet with her almost expressionless face one was forcibly reminded of Paralysis Agitans in an early stage.

*Cranial Nerves.*—Smell, taste, hearing, and sight were all good. There was no contraction of the visual fields, and the optic discs were normal. The pupils were equal, of moderate size, and reacted normally to light and on accommodation. There

were no ocular palsies, and the eyes moved steadily and quickly in all directions. The tongue could be protruded fully, and was central; its lateral movements were also fully performed, although with some awkwardness; when asked to "waggle" her tongue rapidly from side to side, she completely failed to do so, the resultant movements being slow and exceedingly awkward, and taking about a second to complete in each direction, whereas normally the number of such lateral movements possible is about five a second. In other words, the tongue was very spastic.

There was no muscular atrophy anywhere, and in general the muscles were well developed. Nowhere was any movement completely lost, nor could any difference at any time be made out between the two sides, but there seemed to be a definite general weakness of all the movements of the limbs and body, but perhaps more marked in the extremities of the limbs than in the proximal groups of muscles. In all cases the weakness was a slight one, and was accompanied with slowness and some stiffness of movement, although the joints nowhere showed any lesion. At no time was any tremor observed, either during movement or when at rest. There was no inco-ordination of movement anywhere.

There were no subjective or objective sensory disturbances.

*Reflexes—*

Jaw-jerk exaggerated.

Supinator and other arm-jerks all increased, but equal on the two sides.

Knee-jerks equal, brisk.

Ankle-jerks equal, brisk, no clonus.

Abdominal absent.

Sphincters normal.

The plantar reflexes showed that condition which has elsewhere been described by one of us under the term "pyramidal equilibrium"—that is to say, when tested with the limb in the generally flexed position, the plantar response was invariably flexor, but when tested with the leg and thigh in the extended position, the stimulus being applied to the outer edge of the sole, then the plantar response was invariably extensor at first. On repeating the stimulation at short intervals, the third or fourth response and all subsequent ones were flexor, the extensor response having apparently been exhausted. After a few minutes' interval, fresh stimulation would again produce an extensor response.

There were no trophic disturbances.

*Circulatory System.*—The pulse-rate was 86, pulse regular, medium tension, no thickening of the vessel wall. The heart was apparently normal.

The urine was normal, containing no albumin.

She remained in hospital for four months, during which time no marked changes occurred. It appeared as though the stiffness slightly increased, but at no time did any marked weakness come on. The reflexes remained the same.

On November 23, whilst having her breakfast, she suddenly fell back in bed and died almost immediately. She had had no previous attack of angina.

*Diagnosis.*—The chief symptoms of which the patient complained were "hysterical" attacks of laughing and crying, and difficulty in walking so far as she had been accustomed to do. These signs, taken in conjunction with her obvious deficiency of mental concentration, strongly suggested that the whole condition was a functional one. It was only on careful and repeated examination that one could be certain that the movements of the face and limbs were definitely abnormal. The difficulty chiefly arose in that the condition was perfectly symmetrical, so that no differences could be noted between the two sides, and also in that in no single situation was there anything approaching a complete lesion. The stiffness of the facial and other movements then suggested a diagnosis of paralysis agitans, but against this decision was the fact that there were present none of the usual concomitant signs, such as restlessness, sensations of heat, pains in the limbs, etc.; the fact that tremors were absent was hardly of sufficient importance to weigh in the diagnosis.

One sign of spasticity, however, was found which is not present in paralysis agitans, and which, in our opinion, was sufficient in itself to negative a diagnosis of any disease in which the pyramidal tracts are not affected. On several occasions the plantar reflex was tested with great care, and always with the same result, an easily exhaustible extensor response being obtained in the extended position of the limb. It is true that the tendon reflexes all over the body were brisk, and this was useful confirmatory evidence of the spasticity; but the degree of exaggeration of the knee-jerks, for instance, was not greater than is frequently seen in cases of neurasthenia and other functional

conditions. Further, the superficial reflexes of the epigastrium and abdomen were absent. Now it has appeared to us that in cases where the tendon-jerks are increased and the superficial reflexes of the abdomen are also increased, the condition is likely to be a functional one: whereas, if with increased knee-jerks the abdominal reflexes are absent, a diagnosis of spasticity is the more likely. Therefore these signs in this case, confirming the evidence of spasticity as suggested by the plantar reflexes, led to a diagnosis of organic disease involving the pyramidal tracts symmetrically.

If, now, the spasticity of the legs was dependent upon this cause, it seemed very probable that the awkwardness and stiffness of the movements of the face, tongue, and arms were dependent on the same cause, and that the patient was suffering from a generalised spasticity of all her motor functions. There were no signs or symptoms suggesting that any of the sensory tracts were involved, nor were there any signs of nuclear or infranuclear lesions. These facts, together with the history of the onset and the age of the patient, seemed to indicate that the condition was not disseminate sclerosis. The gradual onset with steady progression led to the diagnosis that the disease was a degeneration occurring in the upper motor neurones, and probably not affecting any other of the main neuronic systems of the central nervous system.

An autopsy was made twenty-four hours after death, when rigor mortis was still present. The body was well-nourished and contained abundant subcutaneous fat. The cranial bones, vertebral column, and meninges were healthy. The brain and spinal cord, removed together, showed no obvious signs of disease, but all the large and medium-sized arteries supplying the former were extensively diseased, the vertebral and carotid being specially affected. The smaller vessels running in the pia mater over the surface of the hemispheres were the seat of numerous atheromatous patches. There was no evidence of thrombosis on the surface, and the brain was left to harden before it was cut for further examination.

The lungs were healthy except for an old scar at one apex. The heart was moderately hypertrophied, but the cardiac muscle showed no obvious degenerative change to the naked eye. The coronary arteries were extensively diseased both at their point of

origin and throughout their course. There was some sclerosis of the mitral and aortic valves, and also of the first part of the aorta. The left radial artery did not present any signs of disease.

The liver and spleen were healthy. The kidneys showed little or no loss of cortex, and their capsules stripped readily. The pelvic organs were normal. The thyroid, pancreas, and adrenals were preserved for microscopical examination.

*After hardening* in formalin, the brain was cut in longitudinal horizontal sections, but with negative results as far as any growth, softening, or hæmorrhage was concerned. On the other hand, these sections were remarkable for the appearance produced by the disease of the penetrating vessels. The large majority of the arteries, even the smallest, were conspicuous on account of the rigidity of their walls, and the branches of the perforating arteries on account of a small patch of discoloration which was often present in the tissue surrounding them. This gave a somewhat speckled look to the sections through the internal capsules and basal ganglia.

*Microscopical.*—Sections were cut from the Rolandic cortex and from the internal capsule and stained with logwood and eosin. They confirmed the naked-eye observations in revealing very extensive disease of small arterioles with practically no change in the surrounding tissues. The internal and middle coats of the vessels were both affected as a rule, and in many cases the adventitia contained an abundance of small round cells. The condition of the ganglion cells of the cortex was on the whole satisfactory, and there was an absence of anything like chromatolytic changes. On the other hand many of the cells did not appear to be as large as usual, and it was doubtful whether the number of Betz cells was up to the average.

Sections of a vertebral artery showed marked thickening of the intima, with degenerative changes in the media and small-celled infiltration of the adventitia.

Sections taken from the internal capsule, the pons, the medulla, and from various levels of the cord, were stained by the Weigert-Pal method, and all these were remarkable for a definite sclerosis of the pyramidal tracts throughout their length, and for the healthy condition of most other systems. In the accompanying photograph it will be noticed that the fronto-pontine fibres of the internal capsule are degenerated along with those of motor

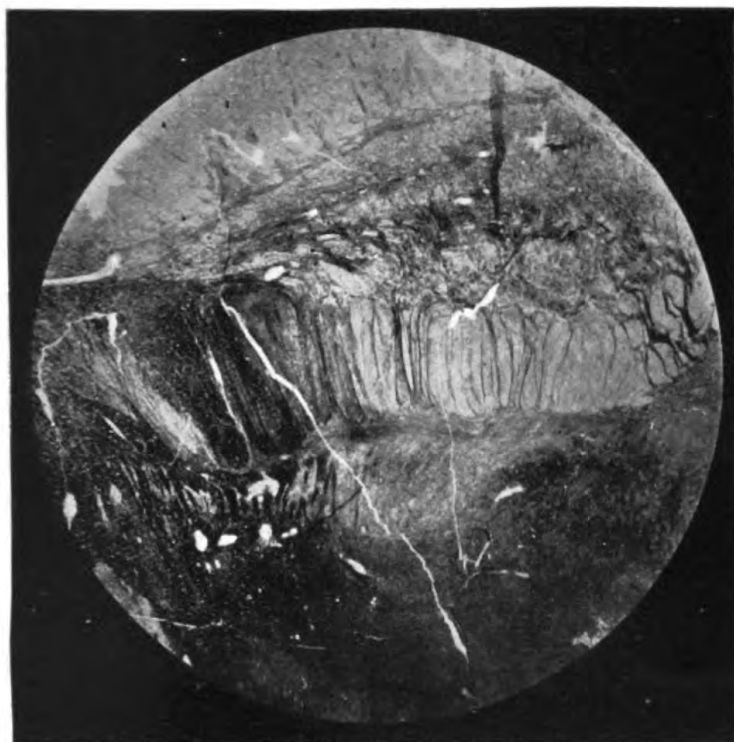


FIG. 1.

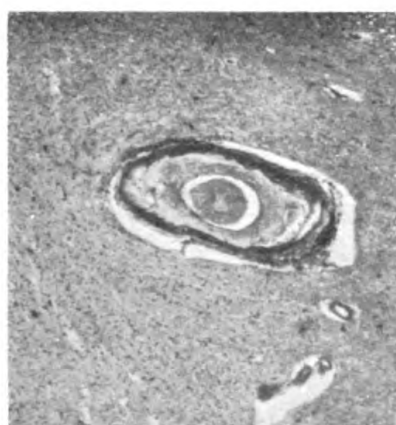


FIG. 2.



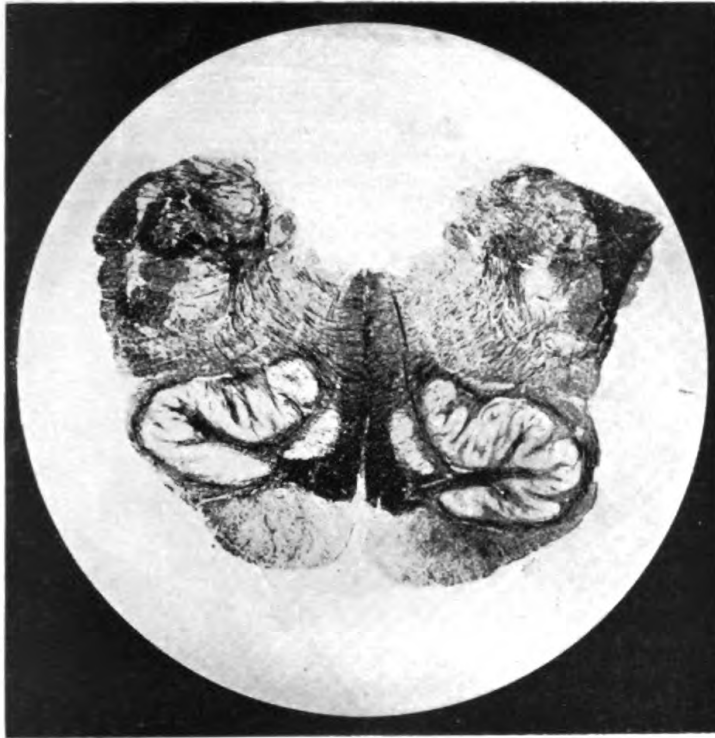


FIG. 3.



FIG. 4.



FIG. 5.



FIG. 6.





origin, leaving only the posterior third of the capsule intact. The degeneration of the crossed pyramids could be traced down to the sacral cord, but the direct pyramidal tracts were not so markedly affected in sections stained by this method.

Sections stained by the Busch method confirmed the presence of degeneration in the pyramidal system and enabled one to observe it in scattered fibres throughout the direct tracts as well. No other definite systemic degeneration was observed by this method, although other parts of the central nervous system presented here and there a few degenerated fibres.

Nissl preparations of the cervical cord presented no abnormal conditions in the ganglion cells of the grey matter.

Sections of the thyroid gland were examined by Dr Chalmers Watson, who reported advanced interstitial changes, marked proliferation of epithelium, extensive disease and calcification of the middle coats of the vessels, and numerous areas of, mainly, lymphocytic accumulations.

The consideration of the morbid anatomy of this case forces us to the conclusion that the condition was a progressive degeneration of the upper motor neurons produced by a failure in the supply of nutrition to the central portions of those neurons, and that this failure of supply was due to the unusually extensive disease of the middle cerebral arteries and their numerous branches.

We are bound to admit that equally extensive vascular disease is occasionally met with in cases in which one or more "strokes" have been followed by a fatal cerebral hæmorrhage or thrombosis, and it is difficult to explain why one person should experience recurring attacks of hemiplegia and another suffer from the chronic progressive ailment of which our case is an example. At the same time it is conceivable that if the accident of a gross vascular lesion is escaped in the early stages of a slowly progressive cerebral arterio-sclerosis, the inactive and vegetative character of life enforced upon the patient may to some extent act as a safeguard against the more violent expressions of the disease.

It may be worth while to point out that the degeneration of the upper motor neurons in our case differs from that which obtains in certain other diseases in which the same system is affected. In Friedreich's ataxy, in subacute combined sclerosis,

and in lathyrism, for instance, the degeneration of the pyramids is not seen much above the lower end of the medulla, while in our case it extends at least as high as the internal capsule. In the former diseases there is probably a gradual decay of the peripheral portions of the neurons; in the latter condition a true, if slow, Wallerian degeneration in a centrifugal direction.

If we were obliged to locate exactly the particular level at which the starvation-process affects the neuron, we should be inclined to select that part of the fibre which lies between the pyramidal cell and the internal capsule, and this for two reasons. In the first place the cortical grey matter was not obviously shrunken nor histologically much altered. In the second place, the centrum ovale is more poorly supplied with blood than the parts lying immediately above or below it.

*Etiology.*—Traumatism was the only etiological factor which could be discovered clinically, but it is clear from the post-mortem findings that the essential factor in the causation was the intense cerebral arterio-sclerosis; it is remarkable that although arterio-sclerosis was specifically examined for clinically, yet no sign of its presence could be detected in the palpable systemic arteries or the retinal arteries, whilst the heart gave no clue to the condition.

*Frequency.*—From the small number of cases verified by post-mortem examination and placed upon record, it would appear that this disease is very uncommon. A diagnosis of "primary lateral sclerosis" used to be very common in the wards of our hospitals, but more careful methods of examination and a wider knowledge of the symptomatology have shown that a large proportion of these cases were in reality disseminate sclerosis. At the same time, it is probable that chronic progressive double hemiplegia occurs with moderate frequency. During the last four years we have both seen several cases in which this diagnosis was made clinically, but in none of them was a post-mortem examination obtained. As compared with disseminate sclerosis, it is probable that the neuronc degeneration of the pyramidal tracts is rare, not more than one case of the latter occurring for forty of the former in hospital practice. In suggesting these numbers, all cases of pyramidal degeneration in which the lower motor neurones are also affected have been excluded, for it seems that the conditions are widely different

in etiology and course; amyotrophic lateral sclerosis tends to run its course in about two years, and the more the spasticity, the more acute the disease; whereas the cases of progressive double hemiplegia are always very chronic, and as in one of the cases seen recently, may still be living in fair general health twenty years after the onset of the disease.

*Nomenclature.*—The term “primary spastic paraplegia” has been rejected in that, being a clinical term, it does not sufficiently define the disease, and also because so many of the cases called by this term in the past have turned out to be merely early stages of some other disease, that the retention of the term is only likely to lead to further confusion. Again, the word “paraplegia” is usually taken to mean a paralysis of the lower extremities, whereas in the condition under discussion, all the muscles of the body, including those supplied by the cranial nerves, become weak and stiff.

“Primary lateral sclerosis,” though a pathological term, is only a partially diagnostic one, and in any case does not express that the degeneration is of the neuron type; it is a term which might usefully indicate the anatomical deduction of certain clinical signs, but is not sufficiently definitive as a name for a disease. And further, in the case we are recording, the condition is evidently not a primary one, but is secondary to the vascular disease.

#### DESCRIPTION OF FIGURES.

FIG. 1.—Section through internal capsule, showing sclerosis of its anterior two-thirds.

FIG. 2.—A section from the neighbourhood of the basal ganglia, showing the diseased condition of the small branches of the perforating arteries.

FIG. 3.—Sclerosis of the pyramids in the medulla.

FIGS. 4, 5, and 6.—Bilateral sclerosis of the crossed pyramidal tracts in the cord.

---

**A CASE OF MUSCULAR DYSTROPHY AFFECTING  
HANDS AND FEET: DEPRESSION AFTER EX-  
HAUSTION, WITH RECOVERY.**

By C. MACFIE CAMPBELL, M.B., Ch.B.

THE following observation is that of a Russian Jewess, who, at the age of 31, after a period of privation and ill-health, developed a condition of depression which made it necessary for her to be committed for treatment to a hospital for the insane. On physical examination, patient presented a well-marked muscular dystrophy which dated back to infancy. The psychosis seemed to have no relation to the muscular condition; both the dystrophy and the psychosis presented points of interest; to avoid confusion the two conditions will be discussed separately at the risk of some repetition.

Several varieties of muscular dystrophy have been described, but the various groups are not now regarded as different conditions, but rather as the same condition with different topographical distribution; transition forms and combinations of the various types are known. The muscular dystrophies as a whole, however, form a distinct group, opposed to the muscular atrophies of nervous origin. The main characteristics of the former are that they usually have their onset in childhood, frequently affect several members of a family, and are very slowly progressive. The dystrophy is usually symmetrical, begins as a rule with the girdles and the proximal muscle groups of the limbs, and only at a later stage, if at all, affects the muscles of the hands and feet; fibrillary contractions are extremely rare, and there is no reaction of degeneration.

DÉJERINE, in his "Sémiologie du Système Nerveux," considers the topographical distribution of the myopathy a most important element in the diagnosis, but this is not an infallible guide; and he refers to the observation by OPPENHEIM and CASSIRER (1898) of a woman, aged 42, with a muscular dystrophy which developed like a case of muscular atrophy of the ARAN-DUCHENNE type. DÉJERINE and THOMAS lately communicated (*Rev. Neur.*, Dec. 30, 1904) the case of a woman in whom the first signs of weakness of the hand commenced in 1868 at the age of 49. In 1893 she showed a muscular atrophy of the ARAN-DUCHENNE type, which

was confined to the upper extremities: fibrillary contractions were present, but no reaction of degeneration. Patient died in 1899, and microscopical examination demonstrated that the nervous system was intact and that the case was one of muscular dystrophy. The following observation is of interest on account of the extreme degree of atrophy of the intrinsic muscles of hands and feet, a distribution which has repeated itself in three members of the same family; while a weakness of the leg muscles, common to these three, has already shown itself in two members of a third generation.

Mrs L. S., æt. 31; Hebrew; admitted March 23, 1905.

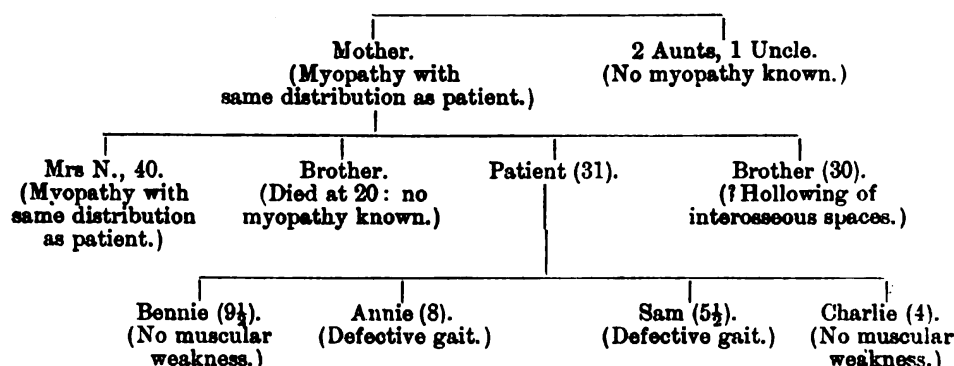
*Family History.*—No information as to grandparents. The father died at 50, cause unknown. The mother is said to have had marked atrophy of the muscles of the hands and a gait similar to the gait of the patient. The muscular atrophy dated from her infancy, and was not progressive so far as her children know. She was able to do her housework until she died at the age of 45, cause unknown. A maternal uncle and two aunts are both said to have been quite free from muscular atrophy. Patient is one of a family of four. One brother had some obscure complaint, dating from childhood, and died at the age of 20. It was impossible to form any clear idea of the nature of his sickness. One brother (30) is alive and healthy; there is some hollowing of the interosseous spaces of both hands, which he himself has noticed, but no weakness of any of the hand muscles can be made out. He has three young children; all seem normal (personally observed).

Patient's sister, Mrs N. (40), has a marked muscular dystrophy with a distribution similar to that of the patient; in both hands the muscles of the thenar eminence show extreme wasting; the short flexor of the thumb seems to have disappeared completely; the hypothenar eminence is considerably atrophied; the interossei are not so much affected; she gives a fair hand grip. The muscles of the shoulder girdle are satisfactory and act well; on the left side, however, the scapula has a slight tendency to leave the chest wall. Both feet are extremely limp and flabby, and assume the talipes position when unsupported; the sole is much hollowed out; the foot can be manipulated with abnormal ease. She cannot dorsiflex the foot at all, and can only very feebly flex it. Flexion of the toes is extremely weak, and there seems to be no power of extension of the toes when that movement is especially tested; when walking, however, she is seen to extend the toes somewhat. The gait is slow and ungainly, the feet (especially the right) drooping outwards as she lifts them up, and therefore having to be raised rather high. She is unable to walk any distance without feeling pains in her legs. Notwithstanding this muscular condition, Mrs N. says that she is in good health and able to do her own housework. She has borne nine children, none of whom show signs of muscular abnormality (5 were examined personally).

The patient herself has four children; all are "double-jointed" in their fingers. Bennie (9½) shows no muscular weakness or atrophy, nor peculiar

gait. *Annie* (8) has a peculiar gait, the left foot tending to rub along the floor; to obviate this she usually raises the left foot rather higher than the right, which causes a slightly swaying gait. No marked weakness of the muscles is made out in testing extension and flexion of feet and toes; the calf muscles are firm and show neither hypertrophy nor atrophy; there is no noticeable weakness in the hand muscles. *Sam* (5½) has a peculiar gait, the left foot being raised high and slightly everted. On the left side dorsiflexion of the foot is weaker than on the right. No weakness of the hand muscles can be demonstrated. On the only occasion on which the electrical reactions were tested, the short flexor of the thumb on the left side gave a slight reaction to the faradic current, while that on the right gave none even with a strong current. The first interosseous muscle on both sides failed to react to a faradic current which gave a marked contraction on an adult. The first interosseous muscle on both sides gave K.C.C. > A.C.C. *Charlie* (4) is said to fall more frequently than other children of his age, but no peculiarity of gait nor muscular defect could be made out.

The family history is shown in the following family tree:—



Patient says that her own muscular condition, like that of her sister, is congenital, and has shown no change until about eight years ago, when the little finger of her right hand began to be slightly flexed. Notwithstanding the condition of her feet and hands, she was able as a child to join in the games of her comrades; she worked well at home and as a domestic, and has been an efficient housewife until the development of her present psychosis.

*Physical State on Admission* (March 23, 1905).—Weight, 73½ lbs.; height, 4 ft. 8½ ins. Patient presents no other malformation than that depending on her muscular condition; she is extremely emaciated, but not anæmic. Her muscular condition is as follows:—

*Superior Extremities.*—The hand is of the simian type, the thumb lying parallel with the other fingers, and the thenar eminence showing extreme atrophy. When the thumb is abducted there is a marked groove on either side of the tendon of the flexor pollicis longus. The short flexor of the thumb seems to have completely disappeared, but an occasional flicker shows that some of the outer head still persists. No action of the short flexor can be made out. Patient is able to oppose the thumb to the little finger, but the slightest touch is sufficient to separate the two fingers. The palm of the

hand is hollowed out, and the heads of the metacarpals, especially that of the index finger, are very prominent, while the tendons of the flexor sublimis digitorum stand out in relief. When the hand is at rest the small finger, and to a less degree the ring finger, are flexed at the first interphalangeal joint; they can be straightened with very little force, but a slight tenseness of the tendon is felt; when patient extends her fingers to the utmost the little finger remains slightly flexed. There is marked wasting of the interosseous muscles, but patient can separate and bring together her fingers, although with little force; fibrillary contractions are occasionally seen. The hypothenar eminence shows less atrophy than the thenar eminence. No reaction to the faradic or galvanic current is obtained from the intrinsic muscles of the hand. The muscles of the forearm and upper arm show a certain reduction in bulk; fibrillary twitching is occasionally observed in these muscles. The muscles are not all equally affected, the deltoid being better preserved and firmer than the biceps, the biceps than the triceps. Most of the muscles show very marked irritability on percussion, especially the deltoid, in which muscle fibrillary twitchings are very prominent. The muscles all act well in proportion to their bulk, and give normal electrical reactions. The condition is practically symmetrical on the two sides. The muscles of the shoulder girdle are all reduced in bulk, but act well; all show marked irritability on percussion, especially the trapezius.

*Inferior Extremities.*—Both feet are in the talipes position owing to the weakness of the muscles of the anterior compartment of the leg. The sole of the foot is flabby and hollowed out; the heads of the metatarsals, especially of the first, are very prominent; the first phalanges are dorsiflexed, while the others are in flexion; the foot is thus slightly similar in appearance to that seen in Friedreich's ataxia. On the right side the big toe is adducted and underneath the second toe. The foot is extremely lax and easily manipulated; the least pressure corrects the deformity, but the foot cannot be brought completely to a right angle with the leg owing to slight retraction of the tendo Achillis; this is slightly more marked on the right than on the left side. The two feet present practically the same condition.

On both sides the muscles of the anterior tibio-fibular compartment show considerable atrophy. There seems to be no action of the tibialis anticus on the left side, and only very slight on the right side. The extensor longus digitorum acts feebly on both sides, while the extensor proprius hallucis acts well on the left side but feebly on the right side. These muscles show no irritability on percussion, and give no reaction to the faradic current. The extensor longus digitorum on the left side gives the reaction of degeneration, while the other muscles of the same group react normally to the galvanic current. The other muscles of the lower extremity are very flabby and reduced in bulk, but act well in proportion to the amount of tissue; they show marked irritability on percussion, and fibrillary twitching is sometimes very well marked; some of the muscles do not react to the faradic current; the condition on the two sides is practically the same. The gluteal muscles are considerably reduced, show very marked irritability on percussion, fibrillary twitching and fascicular contractions.

Patient's gait is slow and hesitating, with marked difficulty in turning; as



each foot is raised it droops and turns outward, the toes are slightly extended as the foot is advanced, the ball of the big toe often rubs along the floor, the foot is put down in a limp way without any spring.

The abdominal muscles show no weakness. There is a flattening over the jaw on each side which suggests some atrophy of the masseter; no weakness of this muscle can be made out; no definite weakness of the muscles of the face can be demonstrated, but patient shows her teeth in a stiff and constrained way. With regard to fibrillary twitching, one may repeat that it is most marked in the muscles of the shoulder and pelvic girdles and of the proximal segments of the limbs. Often there is a continual flickering over the surface of these muscles, especially after exercise.

There is no disorder of sensibility, the patellar reflexes are active and equal; nothing of interest in the thoracic and abdominal organs. Patient received massage and tonic treatment, and during the summer gradually recovered her mental and bodily health. In November her weight had risen from 73½ lb. to 99 lb., the circumference of forearm from 18.3 cm. had increased to 20.2 cm.; while the maximum circumference of the calf, previously 23.2 cm., was now 28 cm. The atrophy of hands and feet and the condition of the muscles of the anterior tibio-fibular compartment were unchanged. On both sides the flexor longus digitorum gave the reaction of degeneration and failed to react to the faradic current. Flexion of the feet was fair, but dorsiflexion almost impossible. The other muscles of the body had recovered their bulk and tone and lost their irritability on percussion, and it was not possible to say that there was any definite atrophy of these muscles; the flattening over the jaw still persisted.

The main points of interest in the above case are the extreme atrophy of the muscles of the hands and feet, the presence of fibrillary twitching in the interossei and short flexor of the thumb, and the reaction of degeneration in two of the atrophied muscles of the lower extremities; the atrophy has presented itself in three generations, but in the two members of the third generation affected it is more limited than in the two previous generations.

*Onset of psychosis at the age of 31, after severe hæmorrhage (Sept. 1904) and months of privation; sleepless, without appetite, thought she would die, suspicious and resentful, attempted suicide (Feb. 1905). On admission (March), quiet, depressed, slow in spontaneous movements, variable in reaction to orders and questions, frequently mute, slightly apprehensive and suspicious, no delusions nor hallucinations, no insight. Improvement during summer; responded more freely, gained insight. November, apparently normal; denied memory of some events during psychosis; return of menstruation.*



FIG. 1.



FIG. 2.



There is nothing of interest in the family history except the myopathic heredity described above ; the description of the brother's sickness given by his relatives baffled diagnosis. Patient was born in Poland in 1873 or 1874 ; her father was a poor tailor and unable to send patient to school. She learned at home to read a little Yiddish ; she was apparently of normal intelligence, efficient in housework. Menstruation came on at fifteen without any disturbance ; she worked as a domestic from the age of ten until she married in 1894 ; she came to New York with her husband in 1895. Her married life was on the whole rather a struggle, and after the first year or so there was considerable household friction ; patient seems to have been of a scolding disposition. She bore four children : Benjamin in 1896, Annie in 1897, Sam in 1899, Charlie in 1901. She nursed her children for four months, two years, one and a half years, and two years respectively. Patient was very temperate in her habits. During the summer of 1904 her husband was sick and unable to work and patient had very insufficient food.

*Onset of Psychosis.*—In the middle of September 1904, patient, who was two months pregnant, began to have slight hæmorrhage ; this persisted for two weeks, and then one night she had a profuse hæmorrhage. A doctor was summoned and removed a large amount of clot ; the hæmorrhage persisted for a week. On the night of the profuse hæmorrhage, according to patient's later account, "I began to speak—not from my thoughts ; when the blood came I got weak, and immediately began to speak other words." She denies having had any hallucinations. Her relatives cannot give any account of any strange talk that night. Patient says that on the following morning she was quite sensible again, but had severe headache for a week. During the next few months patient was quiet, apathetic, and silent. She ate little, was sleepless, said that she was dizzy, and that something ran round in her head. She did a little housework, but sat brooding most of the time. She attended a dispensary and was treated with electricity for her insomnia. Frequently she reproached her husband bitterly for not supporting her better, and showed a suspicious attitude towards him and her relatives. When her husband urged her to accompany him to a doctor, she accused him of paying the doctor to poison her. On one occasion she tore up a prescription, saying that it was for poison. She does not give a satisfactory account of these incidents, and denies such thoughts—"Who would poison another?" According to her retrospective account of this period, she felt that her mind was becoming affected : "Sick and insane is not good, and it was against my will ; I tried to hold it back, twice a week I went to a dispensary . . . my head was weak, my thoughts were feeble, too feeble for you to understand." She was as affectionate as usual to the children, but felt that she must die and that another wife would rejoice in her children ; she rarely spoke to her friends, and then resentfully and suspiciously. On February 1 she was found at night by an open window, and struggled when she was drawn back ; she said that she was warm and had mistaken it for a door ; she now admits that in her anger (*Zorn*) she wished to throw herself out. Next day she was taken to a hospital in Brooklyn ; she scolded her husband vigorously because he was unwilling to pay for a carriage. In hospital she was quiet and depressed, sleepless, and wandering aimlessly about ; at times she heard a roaring sound

in her ears. When visited she showed her friends the same resentful behaviour, turning away her head when greeted, answering curtly if at all. She was discharged March 19 on account of her mental condition ; was unwilling to leave and apprehensive of what was going to befall her ; she lamented loudly. Next day she was taken to the psychopathic pavilion, Bellevue Hospital, where she continued to maintain the same behaviour. She there said : "I am entirely buried—I was buried when I had all the trouble." (She can give no explanation of this statement, which she does not remember.)

She was committed to Manhattan State Hospital, and admitted on March 23 to the Clinical Service of the Pathological Institute.

*Physical State on Admission.*—Patient was much emaciated, but not anæmic—Hb. 100 per cent. (Sahli), R.B.C. 5,280,000 (twice examined). There were no anatomical stigmata and no scars to indicate previous disease. The tongue was moist and coated, showed no tremor. The condition of the internal organs was satisfactory. No information as to subjective feelings could be obtained from patient ; the cranial nerves showed nothing abnormal, there was no sensory disorder, and the only motor disorder was that due to the myopathy described above. The urine contained no abnormal constituents : Sp. G. 1019, T. 98·6°.

*Mental Status.*—On admission, patient was quiet, obeyed requests as a rule, sometimes mildly resisted. When nurse asked her name she replied, "My name is no good." She lay in bed with a slightly worried, woebegone look, occasionally sighed and changed her position. She made no spontaneous remarks, and at first gave no answer to physician's questions. When urged, she answered a few questions, occasionally beginning an answer and not finishing it. A long pause frequently preceded her answer.

Are you sick ? "I have been sick, now I am healthy ; let me go !"

How old are you ? "Thirty-two" (correct).

Where were you born ? "In—in—in—" (sighs).

Where were you born ? "In Europe—in—."

Although apparently very much depressed she did not admit it, said, "I feel good." She allowed her chest to be examined only after asking the physician what he wished to do. When asked to give the right hand she said, "What for?" and did not give it.

She gave very few responses to questions about her sickness ; her grasp of the environment could not be determined, as she repeated the question without answering ; she denied that she was insane.

During the next few days patient continually got out of bed in a slow and deliberate way ; when put back she would resist slightly, but seldom made any remark. She ate and slept satisfactorily. As a rule she sat in bed with the most doleful expression and did not answer the physician's greeting.

On March 25 she refused to enter into conversation with her visitors, would not take some fruit ; when it was pressed upon her she took it from their hands, laid it aside, said, "Lay it down, I'll eat it later."

On March 26 she seemed uneasy and distressed during an interview, squeezed her hands, scratched her head, fumbled with her fingers. She gave very few answers, but these were spoken quickly, although after a pause ;

similarly movements in response to orders were executed with fair speed after an initial pause.

During tedious electrical examinations she complied languidly, only occasionally protesting; she complied with requests, even when she remained mute during a whole interview. Patient was allowed up on April 17, and now sat all day long looking dolefully on the ground. For over a week she had to be dressed by a nurse, and resisted slightly. All her spontaneous movements, *e.g.* at meals, in getting in and out of bed, remained slow.

For several days patient had to be spoon-fed, but during April she ate well, and her weekly weights were 75, 81, 90, 97 lb.

On April 22 she conversed for the first time with her visitors. On two previous visits she had spoken not a word. On May 6 when she saw her child she caressed him, asked many questions about him, talked quickly and eagerly, asked indignantly why the other children had not been brought; she asked her husband to take her home, said that all the other patients were healthy. Although patient talked quickly and with much emotion to her husband and child, she showed the same woebegone, languid behaviour and want of response in an interview with physician on the same day.

In May she refused food for some time, and said that she was not worthy to eat, that she did not work for her food. She now talked a little more readily, denied any knowledge of her environment. "You said you are a doctor. I don't know what you are." She continually refused to accept physician's statement that she was in an insane hospital, but in June she indignantly asked her husband why he had put her in an insane asylum. "I shall never come out alive." As her reason for refusing food, she said that she could not grab food like the others, a quite inadequate explanation. As a matter of fact, patient had to be brought to table, had to be pressed to eat, and usually tried to leave the table before the meal was well begun.

In July she smiled for the first time, said that on admission she had been sick in the stomach, "and I got mixed up in the head." She denied that she had been insane. She quickly, however, gained insight and a good grasp of her environment; she continually importuned the nurses for salts, and stated that she had not had a movement for two or three weeks; her bowels were regular.

In August the hypochondriacal trend had disappeared; she talked freely to physician; took some interest in her environment, knew that it was summer and that she had come in winter. She said that on admission her head was all mixed and that at times she still had this feeling: "All tumbles around; I feel weak in the head."

In November patient was practically well. Menstruation returned on the last day of November, after a year's absence. When the case was reviewed with patient she said of her mutism: "I wanted to speak but couldn't, it was so difficult; when I tried to speak I couldn't open my mouth, I had like a stone on the head." She denied any memory of the trip on the steamer from Bellevue Hospital to Manhattan State Hospital, although in June she remembered it; she denied any memory of her stay in Bellevue, although she remembered going there; although she remembered the various visits of her friends in April, she said that she had completely forgotten the numerous electrical

examinations of her muscles at the same time ; even when the apparatus was again used, she denied that she had ever seen it before. The difference in her memory of the various incidents, if not a mis-statement on her part, possibly depended upon their relation to her interests ; the visits touched her closely, while the examination was merely a bore. This element of personal interest seemed also to explain the marked difference in reaction to a mental examination and to a visit from her child. Patient did not remember refusing food, nor denying that the doctor was a doctor, or the hospital a hospital. She was still slightly confused as to the physicians whom she had known on the service, and she denied to her physician that he was the physician who had examined her previously during her sickness. She maintained that it was another physician, whom she described as being quite similar in every respect. Although patient has completely recovered from her depression, she is at present (December 6) rather loquacious during interviews, and the pleasure which she takes in her environment is rather exaggerated. A convalescent ward is certainly preferable in many ways to her previous quarters in the Ghetto, but she continually refers with enthusiasm to the pleasant environment, the music, the charming nurse, the daily walk, the weekly dose of salts ; she talks with pride of the amount of work she does to help the nurse. Before her menstruation returned she was delighted to have done with it ; on its return she was equally charmed. She is still very bitter against her husband, but her general interests in life seem to be sufficiently normal.

The difficulty of obtaining answers from the patient on admission, and her defective memory or unwillingness to recall details after she had recovered, left several points in the case obscure ; patient denied any memory of her admission into the ward, could on recovery give no account of her mental state on admission, and when confronted with her statement, "this is not a hospital," and with the record of her refusal of food and inadequate excuses, she had either forgotten them or could not be brought to explain them further than, "When one is insane, one says such things."

The most marked features of the case were the depression and general inactivity ; movements were slow and languid, and although her responses were sometimes prompt, they were usually preceded by a pause and frequently were absent altogether. The depression was coloured by suspicion and resentment, which, though of less intensity, were part of her mental equipment before the onset of psychosis. This resentful mood was probably reflected in the negative answers of patient, and in her refusal to accept physician's statements as to the nature of the environments.

In endeavouring to establish the clinical relationships of this

case to other depressions, one is confronted with the great variety of the latter. Some depressions are forms of, or incidents in, other psychoses, alcoholic, epileptic, paralytic, arterio-sclerotic, etc.

Other depressions have a more independent existence. From the great group of these latter depressions several distinct varieties have been separated. KRAEPELIN has endeavoured to give precision to the clinical features of a group of cases occurring usually in the period of involution ("Melancholia" of the involution period). These patients present marked agitation and anxiety and certain delusions of depressing colour, with an otherwise normal stream of mentation. The clinical picture of the present case is quite different from that of this group. Another group of cases has been brought together by WERNICKE under the name "affective melancholia"; the depression in these cases is distinctly secondary to an unaccountable feeling of inadequacy with loss of the ordinary interests of the individual. The patients show little spontaneous activity, but no retardation in response to questions and demands. There is no special tendency to recurrence in such cases, according to WERNICKE.

KRAEPELIN does not allow this as a separate group, but includes it along with certain other recoverable cases of depression in his clinical synthesis of manic-depressive insanity. These patients (depressed phase of manic-depressive insanity) present a primary depression, with blocking of thought and psychomotor retardation; as a rule they recover, but the psychosis tends to recur, the recurrence being liable to take the form of a manic attack. The onset of the attack does not depend upon external causes, but upon some constitutional tendency; external causes may, however, precipitate an attack. In the history of the case recorded above, no history of a previous attack, either of a manic or depressed type, could be elicited, nor was there any trace of any periodic alteration of mood. The exhausting circumstances seem to have acted upon a fairly normal nature and caused a depression which, however, soon overstepped the limit of the normal. The depression, the want of spontaneous activity, the languid responses to questions and orders, were symptoms recalling the depressed phase of manic-depressive insanity; the retardation was not uniform, but was frequently broken through under the influence of her distrustful and resentful mood. Patient acknowledged no feeling of subjective difficulty at first,



said that she was "all right." The professed memory defect on recovery is difficult to understand.

In view of the fact that the psychosis came on after an adequate cause, and that there is no history of any constitutional tendency, one must hesitate to say that this case belongs to the same group as those cases of depression where constitutional tendencies seem all-important and external causes play a merely subsidiary rôle, and of which one characteristic is the tendency to recurrence, with a possible substitution of a manic equivalent for the attack of depression. One can say that patient reacted to exhausting circumstances with a depression of a type similar to one that is found in a certain group of recoverable cases, but one cannot go further and conclude that patient has a constitution which renders her liable to a recurrence of either similar or "equivalent" attacks without adequate cause.

I am indebted to Prof. Adolf Meyer, Director of the Pathological Institute, and to Dr E. C. Dent, Superintendent of Manhattan State Hospital, for permission to publish the above case.

---

## Abstracts

### ANATOMY.

#### THE STRUCTURE OF MOTOR NERVE-ENDINGS IN REPTILES.

(80) (*Sur la structure des plaques motrice chez les reptiles.*)  
AUGUSTIN GEMELLI, from the monastery of Notre Dame des Larmes-Dongo (Lago di Como), July 15, 1905.

A SHORT paper gives a preliminary description of the motor nerve-endings of *lacerta viridis* and *lacerta agilis*. The method of preparation is a modification of Golgi's osmium-bichromate and silver process: pieces of tissue 1 cm. in thickness are placed for half an hour in a mixture of one part of a 1 per cent. osmic acid solution and eight parts of a 3 per cent. bichromate of potassium, to which are added a few drops (from 5 to 10 for each 25 c.c. of the mixture) of a 1 per cent. solution of chemically pure sulphocyanide of potassium. The pieces are next put into Golgi's osmium bichromate mixture for sixty-five to seventy-eight hours, and then transferred to nitrate of silver solution. The passage of the tissue through a mixture of bichromate of potassium and

acetate of copper, as suggested by Golgi, gives equally good results.

This method of procedure is claimed to give results different from those obtained by all previous methods in that only the neurofibrillæ of the nerve and nerve-ending are stained. The fibrils, for the most part, run parallel in the axis-cylinder, and only rarely cross one another; on arriving in the motor end-plate they divide and anastomose freely, forming a very fine network throughout the ramifications of the nerve-ending. Perroncito and other observers had found a system of fibrils in the nerve-endings which were not branches of the medullated fibre, but which formed a second system outside that of the ordinary nerve fibre. Perroncito was unable to trace any connection between the two sets of fibrils. The author finds that very fine fibrils run in the sheath of Henle with the medullated fibre; on entering the end-plate they divide and form a network surrounding the network of the fibrils of the medullated nerve; and the two systems unite with one another by numerous branches.

PERCY T. HERRING.

#### IS THE DECUSSATION OF THE TROCHLEAR NERVE COMPLETE

(81) OR PARTIAL? (Ist die Kreuzung des Trochlearis eine totale oder partielle?) L. BACH, *Centralbl. f. Nervenheilk. u. Psychiat.*, Jan. 1, 1906, p. 16.

BACH has previously published his observations that in rabbits, cats, and apes, and probably also in man, all fibres which spring from the one trochlear nucleus do not decussate in the anterior medullary velum, but that a certain number run forwards with the dorsal longitudinal bundle and join the roots of the oculo-motor nerve.

This statement was challenged by Bernheimer, and Bach's reply is contained in the present paper. He cannot, however, urge any new facts in support of his view, except that Siemerling and Boedeker found degenerated cells in both trochlear nuclei in a case where only the one superior oblique muscle was paralysed.

GORDON HOLMES.

#### LA FAISCEAU EN CROCHET (DE RUSSELL) OU FAISCEAU

(82) CÉRÉBELLO-BULBAIRE. A. VAN GEHUCHTEN, *Névrose*, Vol. iii, p. 119.

THIS tract was first described by Risien Russell as a part of the superior cerebellar peduncle which degenerated only on the opposite side after unilateral lesions of the cerebellum. The name

was proposed by him owing to its shape in cross-section, where it forms the dorsal portion of the peduncle. He was unable to determine its origin and described it as joining the fibres of the opposite peduncle after their decussation.

It has since then been studied by Thomas, Probst, and Lewandowsky, but the descriptions of its course by these authors are not in agreement on many points.

As the fibres which compose it, or at least some of them, decussate in the ventral portion of the vermis, Van Gehuchten has studied in rabbits the degenerations which result from complete median section of the cerebellum by Marchi's method. By this experiment complete degeneration of all the decussating fibres of the cerebellum was obtained. Those which form the *faisceau en crochet* run lateralwards, bend round the superior cerebellar peduncle and form a narrow layer on its dorsal surface, lying between it and Gowers' bundle. Having encircled it they form a compact bundle in the angle between the corpus restiforme and the spinal root of the nervus trigeminus at about the level of the facial nerve. Here the bundle divides into two parts: the one turns ventralwards and medialwards across the facial nerve and runs caudalwards in the middle of the substantia reticularis of the medulla as far as the beginning of the spinal cord; the other, which remains more dorsal in position, runs caudalwards in the medial segment of the corpus restiforme, at first between this and descending vestibular root, and when the latter has disappeared it occupies a still more median position and can be followed to the first cervical nerve. Both these tracts diminish in size as they pass caudalwards, owing to the fact that they give off fibres during their course. Those that come from the dorsal tract form internal arcuate fibres which can be followed to the neighbourhood of the ventral tract. The Marchi method cannot with certainty demonstrate their termination, it only reveals the course of the fibres which are provided with a myeline sheath; but it is probable that they end in the motor nuclei of the bulb, either in those of the motor cranial nerves or in the association centres from which ascending and descending tracts spring.

These tracts are composed of descending fibres, which probably spring from the roof-nuclei of the cerebellum, though this has not been yet definitely proved. Nor is it known if all these fibres have decussated in the cerebellum.

The name tractus cerebello-bulbaris is proposed for the bundle.

GORDON HOLMES.

**METHOD OF STAINING THE NEUROGLIA.** (*Procédé de coloration de la névroglie.*) SABRAZÈS et LETESSIER, *Arch. gén. de Méd.*, Dec. 19, 1905.

THE authors describe the following process for staining the neuroglia, which they recommend especially on account of its simplicity, although it is not so selective as the methods of Weigert and Anglade. Fixation should preferably be carried out by injection of 10 per cent. formalin into the brain by way of the orbit, or into the cord by spinal puncture, as soon after death as possible. At the post-mortem examination thin slices of tissue are in this case placed in 95 per cent. alcohol. In other instances the tissues are to be fixed in 10 per cent. formalin, in saturated watery solution of corrosive sublimate, in a mixture of sublimate solution and Flemming's solution, or by means of certain other fixatives. Pieces are imbedded in paraffin. The staining solution consists of basic fuchsine 1 gramme, carbolic acid 5 grammes, absolute alcohol 10 grammes, water up to 100 c.c. The stain is poured upon the sections fixed to the slide, which is then heated over a Bunsen burner until vapour rises. The slide is then allowed to cool a little and the section is rapidly washed with absolute alcohol and cleared by means of a drop of colourless aniline oil, which should be allowed to act in the vicinity of a bright light. After a few seconds the aniline oil is replaced by xylol and the section is mounted in neutral balsam in xylol.

W. FORD ROBERTSON.

**PHYSIOLOGY.**

**SENSORY CONDUCTION IN THE SPINAL CORD.** (*Ueber die Leitung der Sensibilität im Rückenmark.*) MAX ROTHMANN, *Berl. klin. Woch.*, Jan. 1906, Sn. 47 u. 76.

THE first part of this communication gives the results of the author's investigations into the question of the sensory conducting paths in the spinal cord of the dog, but the details of the experiments are not given in this paper. His method was, briefly, that of combined section, *i.e.* first to cut the anterior columns at the level of the first cervical segment; a few weeks later the posterior columns were sectioned a little lower down; and subsequently the lateral columns were divided. The conclusion arrived at is that no one tract can be regarded as the sole conducting path for any of the various forms of sensation, and in particular that conduction is never entirely one-sided in the cord. One path may, probably does, serve normally to a greater extent than the others, but, if injured, its functions can be more or less completely taken over

by one or more different paths. In detail, the tracts which he assigns for the conduction of the various forms of sensation are :—

(1) *Touch*.—Uncrossed posterior columns and crossed anterior columns.

(2) *Pain*.—Lateral columns mainly, anterior columns slightly ; the crossed being more important than the same-sided. Some conduction may also be possible through the grey matter.

(3) *Temperature*.—Not ascertained.

(4) *Pressure*.—Approximately the same as for pain, but no apparent perception after section of all the long paths in the white matter.

(5) *Muscular Sense*.—All three columns of both sides are available. The lateral columns are the most important, the posterior least.

(6) *Sense of Position*.—Anterior columns. Lateral columns.

The second part deals with the applicability of these results to man. As pathological processes practically never cause lesions sufficiently exact from the experimental standpoint, the only comparable cases are a certain number of instances of cord wounds. Review of some of these shows that extensive restitution of primarily destroyed sensibility can occur, the perception of pain and temperature usually returning to a less degree than that of touch, and all are recovered more slowly and less completely than the power of movement. When one lateral column is divided, the other carries stimuli from both sides of the body ; false projection in the cerebral cortex produces the symptom of allocheiria.

The conditions obtaining in man are very similar to those in the higher mammals, the chief difference from the dog being—

(1) *Touch*.—Unsettled whether the two paths are of equal importance.

(2) *Pain*.—The crossed lateral conduction is of still greater importance than the same-sided. The anterior columns have possibly a slight conducting capacity.

(3) *Temperature*.—Conduction is mainly by the anterior part of the crossed lateral column (Gowers' tract). Restoration by means of the same-sided tract very incomplete.

(4) *Pressure*.—Mainly through lateral column of the opposite side. Also by same-sided lateral column, and possibly by anterior and posterior columns.

(5) *Sense of Position*.—Chiefly same-sided, but also crossed. Through anterior columns mainly, also by posterior part of lateral columns.

J. H. HARVEY PIRIE.

**EXPERIMENTAL SECTION OF THE PYRAMIDS IN DOGS AND**

(85) **APES.** (*Experimentelle Pyramidendurchschneidung beim Hunde and Affen.*) ARTUR SCHÜLLER, *Wien. klin. Woch.*, Jan. 18, 1906, p. 57.

*Method.*—One pyramid was exposed from the front and divided above the decussation, but it was found that it could not be totally destroyed without too much injury to neighbouring parts.

Three dogs were operated on. One died from secondary hæmorrhage four days afterwards, the other two remained alive without disturbance of their general well-being for three weeks; they were then killed, and the cord and medulla examined microscopically. In all, the lesion was found entirely limited to one side.

Full details of the condition in the interval are given. The general result was confirmatory of the observations of various previous experimenters, viz. that no disturbance of locomotion was evident. The touch reflex was more active on the side opposed to the injured pyramid, and there were certain motor disturbances most evident on trying a flank-walk. There was no spasticity, but lateral movements at the hip and shoulder—particularly adduction—were impaired. Other disturbances could not be clearly demonstrated.

Two apes were operated on, but in neither was the lesion entirely confined to one pyramid. Both were kept for three weeks in the best of health. The extremities of the side opposite the section were at first quite paralysed, later they were used merely as supports, and finally took part in forward movements of the body, but although not used so frequently or so readily as the other side, even isolated "single movements" (Munk) could be made. As in the dogs, side movements remained particularly affected—no power of adduction at the hip and shoulder returning.

*Conclusion.*—The pyramidal tracts are the sole conducting paths of the impulses for single movements, for the isolated carrying out of which, participation of the motor elements of the cerebral cortex is indispensable.

Section of one pyramid above the decussation produces loss of certain of these movements, most clearly seen in lateral movements such as abduction and adduction of the limbs.

J. H. HARVEY PIRIE.

**EXPERIMENTAL RESEARCHES ON THE DESTRUCTION OF**

(86) **THE HYPOPHYSIS.** (*Sulla distruzione dell' Ipofisi.*) GAETANO FICHERA, *Lo Sperimentale*, Jan. 1906, p. 739.

THE author gives a very complete critical review of the results obtained by previous observers from destruction of the hypophysis.

He explains many of the discordant results obtained by previous observers as having been due to imperfections in their operative methods.

He operated on fowls, as in these animals the hypophysis is notably developed. By his method, of which he gives a lengthy description, the pharyngeal wall is detached from the basis cranii, and the basilar portion of the sphenoid is opened without the wound communicating with the pharyngeal cavity. The operative wound is thus limited to an incision passing through the geniohyoid and mylohyoid muscles, to the partial detachment of the posterior wall of the pharynx, and perforation of the bony basis cranii. The hypophysis was completely destroyed by a thermocautery, and in every case the autopsy was supplemented by a microscopical examination, to make sure that the destruction had been complete. The operation is said to be simple and rapid, and occupies barely 20 minutes.

In four cases the fowls survived a total extirpation of the hypophysis till they were killed some weeks later. The general disturbances manifested after the operation were in no way characteristic, and there were no late symptoms, except that in the young fowls the author thought that there was some retardation of development—a statement made with every reservation. Examination of all the other glandular organs of the body revealed no abnormality.

Study of the normal hypophysis of the fowl shows a complete absence of the cells described by Traina in the dog as nerve cells.

The paper is supplemented by a very complete bibliography of the literature of the hypophysis. F. GOLLA.

#### ON A NEW METHOD OF INSCRIBING TRACINGS ON THE

(87) **REVOLVING CYLINDER.** (Di un nuovo Metodo per inscrivere Grafiche sul cilindro girante.) GIULIO OBICI, *Riv. di Patol. nerv. e ment.*, Dec. 1905, p. 545.

**THE** new method described in this communication is as follows:—The paper is immersed for a few minutes in the following solution: water, 100; nitrate of ammonia, 150; yellow prussiate of potash, 5. It is then, while still damp, placed on the metal cylinder, which is connected with one of the poles of the induced current; while a steel needle, which takes the place of the usual inscribing lever, is connected with the other pole. Every time the current passes, an azure coloured dot or short line is traced by the needle on the white paper. The cylinder should be covered with tin, or preferably aluminium, as the chemicals used leave a deposit on copper, which might in the long-run affect the weight of the cylinder. When the tracing is removed it should be washed in water to remove the surplus nitrate of ammonia. It will be

rendered clearer by placing it in a bath of water acidulated with HCl.

The dots of the tracing will not be equi-distant, for they will depend on the rate of the movement recorded, and one advantage of the method is that they thus reveal very clearly differences in this rate. If the current be made to pass along a fixed needle also, then a line of equi-distant dots should be obtained, and this line will serve to correct any apparent alteration of movement which may really be due to irregularities in the rotation of the cylinder.

If a tuning-fork be used as interrupter, then the duration of the phenomenon investigated will be shown by the number of dots, and so a Deprez' chronograph may be dispensed with.

The apparatus, moreover, admits of the interruptions being made with much greater frequency than is possible with the ordinary arrangements.

Its advantages are especially great when several tracings have to be recorded on one paper, as many of the measurements ordinarily required for the points of departure are rendered unnecessary.

MARGARET DRUMMOND.

## PATHOLOGY.

**AUTOGENIC REGENERATION.** (*Recherches sur la Régénérescence* (88) *autogène.*) G. MARINESCO, *Revue neurolog.*, Dec. 15, 1905, p. 1125.

MARINESCO brings fresh evidence in support of the autogenic regeneration of peripheral nerves. He specially employed Cajal's reduced silver method, preceded or not by ammoniated alcohol. He employed for experiment young or adult animals, and performed sometimes section of the nerve-trunks, sometimes excision, sometimes avulsion from the central end.

Union of the proximal and distal segments is not essential for regeneration of the distal segment. The process of regeneration is as follows: Embryonic cells appear, derived from the nuclei of the neurilemma cells. In the protoplasm of these cells fine granules appear, which later arrange themselves in linear series within the fundamental protoplasm. These granules become more and more impregnated with "argentophile" substance and have some resemblance to a muscle fibril, that is to say, being constituted by coloured particles separated by clear spaces. As the granules increase in density and the clear spaces become coloured, a nerve fibre is gradually evolved, which may be completely dissociated from the common trunk.

No essential difference exists between the regenerative process



in the proximal and distal segments, nor between that in the newborn and in the adult animal. The only difference is in the degree of intensity and rapidity with which new fibrils are formed. For the return of voluntary movements, however, it is essential that the protoplasmic bands and young fibrils in the proximal segment be in contact with those in the distal.

Marinesco holds that the fact of autogenic regeneration reduces the neurone theory to its proper value, without necessarily destroying it: "elle réduit la doctrine des neurones à sa véritable valeur, sans l'ébranler."

PURVES STEWART.

**THE WALLERIAN LAW.** (*La loi de Waller.*) A. VAN GEHUCHTEN, (89) *Le Névrose*, Vol. vii., p. 205.

THE Wallerian law, as originally propounded, contained a positive—that the portion of a nerve separated from its cell degenerates—and a negative statement—the portion connected with its trophic centre does not degenerate. The positive statement has remained practically unchallenged, and has been but little modified, though recent researches have taught us more of its nature. In the first place it has been shown that the loss of excitability of the peripheral end of a sectioned nerve always precedes the anatomical changes that can be demonstrated in it. The latter begins with a swelling and granular appearance of the axis-cylinder, and later, fragmentation of the myeline sheath occurs. These can be regarded as true degenerative changes or as disorganisation of the fibre, but the later proliferation of the nuclei of the sheath of Schwann is not a degenerative process, but is the first attempt at the reorganisation of the nerve. This reorganisation can proceed till an anatomically perfect and an excitable nerve is produced, even though the part remains unconnected with its trophic cell centre. This is autogenous regeneration, the possibility of which Van Gehuchten accepts as an established fact.

The changes, then, which occur in the peripheral portion of a divided nerve do not represent a degenerative process but a vital reaction, which is dependent on the life of the nerve, and occurs only under suitable conditions; it illustrates the potential hyperactivity of normal cell life.

Waller's positive statement can thus be accepted, but the significance of the word degeneration must be altered; and the possibility of autogenous regeneration must be considered.

The negative statement of the law, that the central end does not degenerate, was accepted for long though Mayser and Lorel demonstrated changes in the central end and in their cells of origin. Then Nissl by his new method showed how constant the changes in the cells are, but the results of other workers with

this method have been discordant. Van Gehuchten limits the discussion to the evidence of degeneration that can be revealed by Marchi's method in the central portion of a divided nerve. He has found that there is but little degeneration of the cell end when a nerve is carefully divided, but that degeneration is constantly found when the nerve is torn out. And this degeneration is not, as has been generally assumed, a retrograde or ascending change, but it begins in the neighbourhood of the cell and spreads towards the point of rupture of the nerve. It can consequently not be due to the trauma. Further, it depends on the intensity of the primary changes which injury to the nerve sets up in the cell; this is so slight that the trophic influence of the cell in the fibres is not disturbed if the nerve is merely divided, but it is great enough to destroy this trophic influence if the nerve is roughly torn out. The degeneration of the central end of the nerve sets in later than that of the peripheral end, though both are due to the same cause, i.e. loss of the trophic influence of the cell.

The negative part of the Wallerian doctrine is therefore not true in certain experimental conditions, though the degeneration of nerves is always consecutive to an atrophy or other change of the nerve cells or trophic centres.

GORDON HOLMES.

**A FURTHER CONTRIBUTION TO THE STUDY OF THE ANATOMICAL BASIS OF SYPHILITIC SPINAL PARALYSIS.**  
(90)

(Ein weiterer Beitrag zur Lehre von der anatomischen Grundlage der "syphilitischen Spinalparalyse.") D. H. NONNE,  
*Deut. Zeitsch. f. Nervenheilk.*, H. 5-6, 1906, p. 369.

THE author refers to a former paper of his in which he maintained that the symptom-complex known as "syphilitic spinal paralysis" has its origin in one or other of the following anatomical conditions:—

- (1) A chronic patchy transverse myelitis with ascending and descending degenerations.
- (2) A similar condition combined with a primary pyramidal system degeneration.
- (3) A primary pyramidal system degeneration alone; or
- (4) A combined system degeneration involving the posterior column, the pyramidal system, the direct cerebellar and antero-lateral ascending tracts.

Together with these changes may be found morbid conditions of the meninges and blood-vessels.

A case is then cited which was under observation for 13 years, and finally examined post-mortem. A man, 57 years of age, with a history of syphilis 30 years previously, had for 3 years experienced slight difficulty in gait, slight bladder trouble, and occasional pains

in the legs. His pupils were small and reacted sluggishly to light. The other cranial nerves and the upper extremities were healthy. There was slight spastic paraplegia with hardly perceptible blunting of sensibility in the skin of the lower extremities. The condition remained stationary until 13 years later, when the patient died of pneumonia.

Post-mortem examination revealed a very little patchy chronic myelitis in the dorsal region of the cord without definite secondary degeneration, some degeneration in Goll's tract in the cervical and upper dorsal region, some degeneration in the pyramidal tracts of the lumbar region, general thickening of the blood-vessel walls throughout the cord, chronic endarteritis of the anterior spinal artery, and slight posterior meningitis of the cervical and dorsal regions.

Nonne regards the case as one in which a primary combined system degeneration was associated with a diffuse myelitic affection.

E. FARQUHAR BUZZARD.

#### **THE FIBRILLAR STRUCTURE IN PROGRESSIVE PARALYSIS.**

(91) (Über Fibrillenbilder der progressiven Paralyse.) K. SCHAFER  
(Budapest), *Neurol. Centralbl.*, No. 1, 1906, p. 2.

At the beginning of this article there is a résumé of most of the literature of the subject.

The author's work has been done with Bielschowsky's silver impregnation method. The areas examined were the anterior and posterior central gyri, the paracentral lobule, the operculum, the two frontal convolutions of the convexity, the cortex of sulcus cruciatus (basal frontal), the first temporal, gyrus angularis, first parietal convolution, calcarine cortex, and cornu ammonis.

In a medium sized pyramid of the posterior central gyrus the fibrils of the apical process seem to be isolated, but those of the cell body form a reticulum which possesses more prominent trabeculæ running through, in direct continuity with certain fibrils of the dendrites.

Between these prominent threads there stretch finer and paler ones showing slight swellings at the nodal points. The meshes of the reticulum are round or polygonal, more elongated towards the apical process, and gradually merge into its structure.

In the smaller pyramids of the anterior central sulcus the network stains more faintly towards the apical process. These cells show the following marked changes: swelling of cell body and apical process; disappearance of fibrils around the nucleus, which is deeply stained; the peripheral part of the cell is deeply stained, and here the remains of the network are only recognised with difficulty. The protoplasmic processes are absent.

In progressive paralysis the stages of degeneration are firstly, swelling and deep staining of the nodal points of the reticulum with granularity; the meshes become rounded, the threads of the network disappear, and the star-shaped nodal points are left. These then break up into finer granules.

In the large pyramidal cells of the paracental lobule the dichotomous division of the fibrils running from the apical process into the cell body is well shown; the finer sub-divisions are lost in the reticulum. On comparison with cells from the same area in a normal brain, certain changes are demonstrable. The cell body may be swollen and the apical process diffusely stained, while the reticulum is well preserved; or the cell body may be diffusely stained, while the fibrils are evident elsewhere. Often the interfibrillar substance is deeply stained and the fibrils are disintegrated. To this one can add a terminal form consisting in complete loss of the network.

All the areas examined showed changes. The early changes prevailed most in the central gyri; all the other regions showed a high degree of alteration. In the region of the operculum the disintegration of the reticulum was more marked than in the other parts of the central convolutions.

This widespread alteration of the nerve cells is accounted for by the advanced stage of the disease.

In the anterior cornual cells (cervical) the fibrils of the dendrites run parallel to each other. At the meeting point of dendrites a triangular space is left to accommodate the Nissl bodies.

The fibrils in the processes are massed together, and as they converge on the cell body they form a thick band passing round its margin. In the cell body is a wide-meshed network with star-shaped nodal points.

The author regards the broad dark band where the apical process meets the cell body as a pericellular network with oblique meshes. This reticulum appears to end at the point indicated owing to the cell being cut in section.

The cell is slightly swollen and diffusely stained, constituting what the author describes as incipient change.

More advanced changes are found in the "Strangzellen"; the fibrils in the dendrites are no longer visible and are reduced to granules arranged in rows. The cell body shows similar changes, contains fine granules and certain ring-like structures bound together to form a few meshes (residues). Such alterations are found at all levels of the cord.

DAVID ORR.

**ON THE FUNCTION OF DEGENERATE MUSCLES. SECOND**

(92) **PAPER. TIME OF LATENT EXCITATION.** (*Sulla Funzione dei Muscoli Degenerati. IIª Comunicazione. Tempo di eccitazione latente.*) Dr GUIDO GUERRINI, *Lo Sperimentale*, Nov.-Dec. 1905.

THIS paper gives an account of a series of experiments made by the writer on the muscles of frogs in a state of fatty degeneration, with the view of determining the period of latent excitation, and compares the results obtained with the results of similar experiments on healthy muscles. Full descriptions of the experiments and tables showing the numbers obtained are given. A bibliography is appended.

*Chief results obtained.*—1. The time of latent excitation is extraordinarily long in degenerate muscles as compared with healthy muscles, and increases with the amount of degeneration.

2. Degenerate muscles differ from healthy muscles in that no connection can be discerned: (1) between the weight and the time of latent excitation, or (2) between the time and the intensity of the stimulus, or (3) between the time and the distance between the electrodes, or (4) between the time and the distance between the end of the muscle attached to the lever and the nearer electrode.

3. Fatigue may prolong the period of latent excitation, but the increase is less in proportion than occurs in healthy muscles, and appears more in the opening than in the closing contraction.

4. Variations of temperature between 12 and 17 degrees C. do not affect the results.

The article concludes with a short examination in the light of these results of (1) the hypotheses which have been advanced to explain the phenomenon of latent excitation, and (2) the theories which have been formulated to explain the mechanism of muscular contraction.

MARGARET DRUMMOND.

**MALFORMATION OF THE BRAIN IN HATTERIA PUNCTATA.**

(93) (*Eine Gehirnmissbildung bei Hatteria punctata* [*Sphenodon punctatus*]). ERNST SAUERBECK, *Nova Acta. Abh. d. k. Leop.-Carol. deutschen Akad. d. Naturforscher*, Bd. lxxxv., Nr. 1, 1905, pp. 1-120, 2 plates and 12 text-figures.

THIS is an exhaustive and well-illustrated account of a malformed brain of a Hatteria embryo measuring about 4 cm. from the tip of the nose to the root of the tail. The length of the head, from the tip of the nose to the articulation between the atlas and the occipital bone, was 12 mm. The skin showed a higher grade of development than that of a normal embryo of about the same size.

To the naked eye, the malformation was limited to the head. The trunk and limbs exhibited no abnormality externally, nor did microscopic examination of them reveal anything either teratological or pathological.

The intact head, to the naked eye, showed the following aberrations: (1) There was an evident disturbance in the normal proportion of the mandible to the upper part of the skull. The mandible was large relative to the size of the rest of the head, and also in comparison with the body as a whole. (2) The upper part of the skull was abnormal, since its anterior portion projected upwards instead of curving downwards. As a consequence, the whole of the tongue and the teeth were visible from the exterior. (3) Instead of a bulging in the position of the eye, there was an in-sinking of the lateral wall of the head. (4) On the dorsal aspect of the head, and between its anterior and posterior parts, was an ovoid mass, whose long axis was sagittal. The under surface of the mass was connected with the roof of the cranium by means of a broad pedicle.

The whole embryo, after separation of the head from the trunk, was embedded in paraffin, and sections 20  $\mu$  thick were made of the head in a sagittal direction and of the trunk in a transverse direction. For purposes of comparison, two normal embryos were also sectioned.

Concerning the normal embryos, the author says that the eyes formed almost half of the total mass of the head. The brain showed a typical five-vesicle condition, and was constructed essentially to subserve the purposes of the special senses of hearing, sight, and smell. In accordance with the large size of the eye, the mid-brain formed a very considerable proportion of the whole brain.

It is difficult to convey an adequate idea of the brain of the malformed specimen without illustrations. The figures accompanying the article, however, present a very clear indication of the state of the parts. The author states the characters of the deformity, in general terms, as being (1) the absence of the eyes, and (2) an abnormal development of the brain and skull, such as may be expressed by the word "hemi-exencephaly." Of the whole brain, the medulla alone was contained within the cranium. The cerebellum, the mid-brain, the diencephalon, and the telencephalon were discovered in the ovoid mass on the dorsal surface of the head. This malposition was necessarily accompanied by an elongation of the neural tube as far forwards as the cephalic flexure.

The most striking peculiarity of the specimen was the persistence of a kind of neuropore, out of which parts of the brain had been everted. Or, as the author states, there was a prolapse of the neural tube comparable, in a manner, with a prolapse of the uterus. In addition to the neuropore was the persistence

of another early embryological condition, namely, a direct histological continuity of the wall of the neural tube with the epidermis of the dorsal surface of the head.

The roof of the fourth ventricle, the medulla, the cerebellar lamina, the arch of the mid-brain, the infundibulum, and the floor of the third ventricle were easily recognised as such. But the epiphysis, the roof of the third ventricle, and the optic chiasma were either absent or altered beyond recognition.

There was no development of the nervous part of the eye, nor any trace of the lens. But it is remarkable that sections revealed a rudimentary external eye (cornea) provided with a conjunctival sac and a lachrymal gland. The ocular muscles and their nerves were represented. A considerable proportion of the muscles were fairly clearly differentiated, but it was not easy to identify the abnormal muscles with those of a normal eye.

The condition of the primordial cranium and the etiology of the malformation are discussed.

The communication, according to its sub-title, is a critical monograph designed as a contribution to a rational teratology of the brain; and its substance is, briefly but clearly, summarised in the words: *Eversio encephali e neuroporo. Transgressus persistens laminæ nervosæ in epidermidem. Anophthalmia duplex partialis (defectus oculi nervosi et lentis).*

O. CHARNOCK BRADLEY.

### CLINICAL NEUROLOGY.

**ARSENICAL NEURITIS.** (*Ueber Arsenic Neuritis.*) FRANZ CONZEN, (94) *Neurolog. Centralbl.*, Jan. 2, 1906, p. 18.

UNDER this title the author describes the case of a girl, aged 24 years, who was engaged in sewing and preparing furs. Her previous health was normal. In September 1904 she commenced this work and had to dip the first, second, and third fingers into an arsenical solution. In three months she noticed these fingers easily "went to sleep," and were blanched, especially in cold weather, the thumbs and little fingers being normal. She had no pains in the arms or legs and no digestive or conjunctival trouble. There was found no pigmentation, no loss of power, and no muscular atrophy. The two terminal phalanges of the first, second, and third fingers of both hands were cyanotic, especially the index finger of the right hand. The skin of the fingers is shiny and very smooth. The nails in both longitudinal and transverse directions are strongly furrowed. The nails of the first and second fingers of both hands, and especially the nail of the third finger of the left hand, are thickened and stratified. The colour is a dirty greyish-white. The fingers are very painful

on pressure, tactile sensation is normal, and there is marked hyperalgesia of the finger tips; both hands sweat profusely.

The author considers that in this case there is a local poisoning by arsenic which is taken in through the uninjured skin. A photograph of the affected hands is given.

ERNEST S. REYNOLDS.

**DIPHTHERITIC PARALYSIS OF THE LEFT HYOGLOSSUS.**

(95) (*Diphtherische Lähmung des linken Hyoglossus.*) HAMBURGER, *Wien. klin. Wchnschr.*, December 21, 1905, p. 1370.

HAMBURGER exhibited at the Gesell. f. in. Med. und Kinderheilk. in Wien, a boy suffering from diphtheritic paralysis of the left hyoglossus which was manifested by inability to depress the corresponding side of the tongue on the floor of the mouth. The other tongue muscles were intact, but cycloplegia, paralysis of the palate, and paresis of the legs were present. The case had received antitoxin.

J. D. ROLLESTON.

**FRIEDREICH'S ATAXIA.** WHARTON SINKLER, M.D., *New York Med.*

(96) *Journ.*, 1906, p. 65.

THIS short paper is almost wholly concerned with the clinical aspect of the disease; the few sentences devoted to its pathology are of no importance. The clinical notes of the thirteen cases which the author records are in many respects incomplete and "ataxic," whilst in one or two instances the accuracy of the diagnosis seems open to question.

The main facts, so far as they are presented, may be summarised as follows:—

There was no history of ataxia or allied disease of the nervous system in the patients' parents or among their ancestors. The sex of the patients was about equally divided. The age of the first appearance of the disease varied between 2 and 21 years.

The first symptom observed was usually an unsteady gait, though in one or two cases inco-ordination in the upper limb and hand, or weakness of the back, were noted before the gait seemed affected. With regard to the reflexes, the knee-jerk was absent in nine cases, present in two cases, and markedly increased in two others, whilst the plantar reflex was absent in five cases and present in eight.

Contractures existed in seven cases. Speech was affected in a similar number. With respect to eye symptoms, nystagmus existed in nine cases; vision was as a rule good, but the colour field was contracted in one case; the pupils were practically normal to light



and accommodation in all those cases where an examination was made. In one instance, however, the light reflex was rather slow.

An antero-posterior or lateral spinal curvature was noted in eight cases. In one case the spine was normal; while in four there is no record as to its condition. In two of the cases ataxia seems to have followed a paralytic attack, which overtook one patient at the age of three and the other at the age of five years.

HARRY RAINY.

**POTTS' PARAPLEGIA WITHOUT EITHER LEPTO-MENINGITIS OR COMPRESSION, ETC.** (*Paraplégie pottique par myélomalacie, sans leptoméningite ni compression, etc.*)  
DUPRÉ and CAMUS, *Rev. Neurolog.*, Jan. 15, 1906, p. 1.

A RAPIDLY progressive paraplegia was found post-mortem to have been caused by Potts' disease of the fourth, fifth, and sixth dorsal vertebræ, with a corresponding hypertrophic tuberculous pachymeningitis. There were no meningo-medullary adhesions, no compression of the cord, no lepto-meningitis; on the other hand, at the same level as the rest of the disease was a zone of softening in the cord, with degeneration of the columns of Goll and periarteritis of radicular vessels. Apparently the morbid process had established itself in the cord *via* the posterior roots and by involvement of blood-vessels; the clinical symptoms were therefore due to local myelitis supervening in Potts' disease.

S. A. K. WILSON.

**CEREBELLAR TUMOUR WITH MENTAL SYMPTOMS.** (*Ein Fall von Neubildung des Kleinhirns mit psychischen Symptomen.*)  
KURT BERLINER, *Giessen. Klinik f. psych. u. nerv. Krankheiten*, Bd. 1, H. 1, 1906.

DR BERLINER gives a careful account of a case of cerebellar tumour in which mental symptoms appeared early in the course of the disease, the patient being admitted to the clinic on account of mental excitement and disorderly and violent behaviour. The first symptoms were headache and vomiting. Later the gait became affected, and then visual hallucinations appeared with profound depression. Amongst other signs and symptoms were occipital pain and tenderness, papillitis and retinal hæmorrhage, nystagmus and cerebellar ataxia. A prominent feature was almost continual unrest of the total musculature; the movements not being involuntary, unco-ordinated spasms, for, though not intentional, they could be inhibited, were mentally conditioned, and, in the author's opinion, were imperative in character. The patient

died on the nineteenth day after admission, and a tumour was found abutting into and completely filling the fourth ventricle. At the time of writing no microscopic examination had been made, but the tumour was probably gliomatous. On this account the origin and extent of implication of cerebellum have not been ascertained. Amongst the psychical symptoms the author considers that the visual hallucinations, motor excitement, etc., are most easily explained as the psychic equivalents of epileptic attacks.

R. CUNYNGHAM BROWN.

**CHOREA AND ITS NEURONIC ASPECT.** Sir WILLIAM GOWERS, (99) *Phonographic Record of Clinical Teaching and Med. Sc.*, Aug. and Sept. 1904, Vol. x., Nos. 8 and 9, pp. 113 and 129.

In an article in two numbers of this journal Sir William Gowers amplifies a lecture he had delivered previously at the National Hospital, Queen's Square. Chorea is defined as a "derangement of the nutrition and function of the motor structures of the cerebral cortex, sometimes one-sided only, often on both, and sometimes extending to those structures of the cortex concerned in mental processes." On the old hypothesis that the nerve impulse proceeded from the cell, which served as the battery generating nerve force, a change in structure of these cells was expected in chorea, and it was a matter of surprise that no constant changes were found there. With the rise of the neurone theory, an exposition of which is given with a description of the facts on which it was based, it was recognised, however, that the nerve cell was concerned with maintaining vitality rather than with originating function. The source of the nerve impulse being referred beyond the cell to its dendrites, it is reasonable to suppose that in chorea, which is primarily a defect in co-ordination, the affection may be dendritic. This affection may be one of nutrition as well as of function; irregularity, both in time and extent, of dendritic action would account for most of the motor manifestations of chorea.

The three causal influences that stand out in regard to the disease are: first, its occurrence in childhood at the time when the tendency to excessive motor activity is so pronounced; secondly, the action of powerful emotion, particularly alarm, as probably deranging, through shock, regular dendritic functioning; and last, the altered blood state that the disease shares in common with rheumatic fever.

A series of cases are then described, and the diagnosis between the condition and habit spasm discussed. Two of the cases had chorea gravidarum, and the suggestion is made that in pregnancy the mental state approximates towards that of a child, thus accounting for the frequency with which pregnancy is found in

cases of adult chorea. The association of chorea with the vomiting of pregnancy and with rheumatic endocarditis is dealt with.

As to treatment, Sir William Gowers recommends absolute rest, antimony wine, small doses of chloral and antipyretics. Arsenic has been much overrated, but strychnine is of considerable value towards the end of the attack. Hypodermic medication is to be discountenanced in the disease.

ERNEST JONES.

**CASE OF MULTIFORM TIC, INCLUDING AUTOMATIC SPEECH (100) AND PURPOSIVE MOVEMENTS.** MORTON PRINCE, *Journ. Nerv. Ment. Dis.*, Jan. 1906, p. 29.

THE patient was a man, æt. 35, who presented a combination of various tics. Some consisted of ordinary choreiform movements of the eyelids, face, and arms, while others were more complex purposive movements. Many of these automatic physiological movements were remarkable in themselves, but most unusual were the automatic speech which was interjected in the midst of nearly every sentence he uttered, and certain purposive movements when he handled a razor or a knife. When asked whether he had difficulty in pursuing his occupation, he answered, "Christ, no; hell-nigger, —," in an explosive, jerky way, and then quietly added, "Yes, I have been obliged to give it up." He had naturally a nice mind, was well-mannered, well spoken of, and felt his affliction keenly. He was entirely unaware of what he would say automatically until the words were actually spoken. The patient in addition revealed a slight degree of mental infantilism.

From the point of view of diagnosis, it is very important and significant to note that his tics occurred chiefly when his attention was directed to prevent them, *i.e.* they were not of the nature of mere "absent-minded phenomena."

S. A. K. WILSON.

**FACIAL TIC CURED BY SUGGESTION.** (*Un cas de tic de la face (101) guéri par suggestion.*) IOTYKO, *Journ. de Neurol.*, Jan. 5, 1906, p. 1.

A YOUNG woman, 22 years of age, had suffered for eight years from various and varying facial tics, including rapid blinking or nictitation, throwing back of the head, twisting of the mouth, frowning, and general grimacing. All were clonic, with the exception of the head deviation. She presented in addition a typical mental infantilism. Treatment by the combined methods of enforced immobility (Brissaud), respiratory gymnastics (Pitres), and psychotherapy, as well as by other means, was followed by what promises to be a permanent cure.

S. A. K. WILSON.

**AN UNDESCRIBED SYMPTOM OF PALATAL PARALYSIS.**

(102) (Ein nicht beschriebenes Symptom der Gaumenlähmung.)

SCHLESINGER, *Neurolog. Centralbl.*, Jan. 16, 1906, p. 50.

THIS paper records three cases of palatal paralysis, one accompanying multiple sclerosis, and two present with cerebro-spinal lues. In all three the peculiarity consisted in the fact that in the recumbent position the speech was good or quite unaffected, while as soon as the patient sat up the effect of the paralysis became evident in the speech. This is explained by the passive position taken up by the palate bringing it nearer to the back of the pharynx as the patients lay than when they sat up. In a dozen other cases of palatal paralysis examined by the writer, this peculiarity was not present.

JOHN D. COMRIE.

**BILATERAL CIRCUMSCRIBED FACIAL ATROPHY. (Ein Fall**

(103) von doppelseitige umschriebener Gesichtsatrophie.) ALFRED

SCHLESINGER, *Archiv f. Kinderheilk.*, Bd. 42, H. 5 u. 6, 1905, p. 375.

BILATERAL progressive facial atrophy is much more rare than the unilateral form; of the former, only eight cases are on record; of the latter, more than one hundred. The main features of Schlesinger's case are as follows: The patient was a girl aged 10 years; in her fifth year, after an attack of measles, the skin of both cheeks became thin and discoloured; the wasting extended gradually to the subcutaneous tissues. After progressing steadily for about a year the atrophy ceased, and since then the condition has remained unaltered. The child now presents the following appearance: She is poorly nourished; the circumference of the cranium, which is dolichocephalic, is 56 cm. The hair, frontal region, and orbital ridges display no abnormality. Both cheeks are deeply sunken, the skin is thinned, and on palpating from the mouth there appears to be no fat or connective tissue between the skin and mucous membrane. The muscles of the face are normal. When the cheeks are puffed out the atrophic regions bulge in a balloon-like fashion; on showing the teeth they fall into deep folds. Otherwise the face and cranium are normal in structure, the bones not being involved in the atrophic change. There is facial irritability, but the electrical reactions of the muscles innervated by the seventh nerve are normal. There is, however, a slight degree of narrowing of the right pupil and right palpebral fissure. The noteworthy points of the case are the appearance of circumscribed facial atrophy in a young person after an infectious disease, and the entire absence of any neuralgia, etc., pointing to

implication of the fifth nerve, while there are evidences of sympathetic lesion.

Schlesinger shortly discusses the various theories which have been put forward as to the cause of the condition—trophoneurosis (Romberg), neuritis of the fifth nerve (Virchow, Mendel), lesion of the Gasserian ganglion (Jendrassik), progressive aplasia (Bitot and Lande), local toxin action, bacterial or otherwise, acting on tissues previously weakened by disease (Möbius)—and thinks that his case can be most readily explained on the last of these hypotheses.

J. S. FOWLER.

**THE ABDOMINAL REFLEX IN ENTERIC FEVER.** (I riflessi (104) addominali nell' ileo-tifo.) ORTALI, *Gazz. degli Osped.*, October 15, 1905, p. 1303.

ORTALI investigated the abdominal reflex in 61 cases of enteric fever. In the prodromal stage it was present in 21 out of 22 males and in 36 out of 39 females. During the height of the disease it was completely abolished in 41 severe cases, diminished short of absolute extinction in 20 mild cases. During defervescence the reflex became slowly re-established, until in convalescence it returned to its normal activity. In 3 cases of severe relapse the reflex, which had resumed its normal condition, underwent the same changes as in the primary attack. In 2 cases of recrudescence it became abolished a second time after it had begun to return. The change in the abdominal reflex was not due to the general condition, since the other reflexes, superficial and deep, were not similarly affected. A local cause must therefore be sought. All organic changes in the muscles, nerves, and spinal cord must be excluded because of the prompt return of the reflex during the decline of the disease. The condition must be regarded as due to a functional interruption of the reflex by direct influence of the intestinal lesions on the reflex centre in the thoracic portion of the spinal cord.

Ortali's observations and conclusions coincide with, but were made before, the publication of those of Sicard (*Presse Médicale*, January 11, 1905).

In like manner, prompted by Sicard's paper, but prior to the publication of Ortali's researches, the reviewer carried out investigations which will form the subject of a future communication. His conclusions will be found to confirm and supplement those of his two predecessors.

J. D. ROLLESTON.

**APHASIA, HEMIPARESIS, AND HEMIANÆSTHESIA IN  
(105) MIGRAINE.** JELLIFFE, *New York Medical Journal*, Jan. 6,  
1906, p. 33.

THIS is a detailed account of three cases of migraine presenting unusual accompaniments, though the writer, in commenting upon them, refers to numerous other recorded cases with similar features (six references).

In one case, a man of 38 had suffered from chronic migraine for twenty-three years. The attacks were almost always the same, consisting of a "fortification" scotoma lasting for half an hour, and followed by severe headache. For some time these attacks recurred daily about 11 A.M., but latterly the patient had only monthly or quarterly visitations. In two attacks there was distinct motor aphasia lasting for about five minutes during the stage of the ocular phenomena. One of these occurred at school, the other later in life.

In the second case a musician, aged 35, had suffered from headaches since boyhood. These affected the right side of the head, and the pain even ran down the right arm, and was accompanied by weakness of the right leg. The attacks were followed by numbness and pricking in the right leg and arm, lasting often several days.

In the third case, a youth of 19 was suddenly seized by inability to write, lameness in the right leg, sickness, and difficulty in speaking, followed after some time by intense headache. From this attack he slowly recovered. Subsequently he had another, with scintillating scotoma, dizziness, and sickness, followed after some hours by complete unconsciousness and cardiac irregularity, and later by severe left-sided headache and anæsthesia especially marked on the right side of the body. From this attack, recovery was slow but complete.

JOHN D. COMRIE.

**GENERAL AND ALMOST COMPLETE RETRO-ANTEROGRADE  
(106) AMNESIA, WITH DELUSIONS, ETC., IN A CASE OF  
HYSTERIA.** (Amnésie rétro-antérograde générale et presque  
totale ; délire ; anésthésie considérable des diverses sensibilités  
chez une hystérique.) DELACROIX and SOLAGER, *Rev. Neurolog.*,  
Jan. 15, 1906, p. 6.

THE interest of the case lies in the extraordinary degree of retro-grade amnesia presented by the patient. Nine-tenths of the history she gave of herself turned out to be the purest delusions, so that all she knew of herself and of her past amounted to exceedingly little. At the same time her anterograde amnesia

was evidenced by her inability to learn or understand or assimilate even the simplest facts. She showed unmistakable stigmata of hysteria, associated with widespread changes in different forms of sensation. The authors are unable to say whether the amnesia is to be attributed to this profound alteration in sensation or to enfeeblement of mental synthesis.

S. A. K. WILSON.

**FAMILIAL ORETINISM.** (*Familiärer Kretinismus.*) JAEGER, *Giessen*.  
(107) *Klinik f. psych. u. nerv. Krankheiten*, Bd. 1, H. 1, 1906.

DR JAEGER, after some general observations on cretinism, gives in this paper a very complete account of the four children, two brothers and two sisters, of a couple, of whom the father was a drinker with an alcoholic heredity, and the mother healthy and of sound stock. The father and mother were blood relations and the union unhappy. The oldest child, a girl, is a mild cretin; the second, a boy, a myxoedematous imbecile of exceedingly rudimentary development; in the third, a boy, the cretinism is moderately pronounced, and the fourth is as yet (æ. 12) completely normal in development. The possibility of another father has been carefully excluded, and the author considers these to be cases of sporadic cretinism with, as ætiological factors, parental consanguinity and alcoholism.

R. CUNYNGHAM BROWN.

**MATHEMATICAL ANALYSIS OF FATIGUE CURVES AS AN**  
(108) **AID TO DIAGNOSIS IN NERVOUS DISEASES.** (*L'analyse mathématique des courbes de fatigue comme procédé de diagnostic dans les maladies nerveuses.*) IOTEYKO, *Journ. de Neurol.*, Jan. 5, 1906, p. 7.

THIS paper presents succinctly some of the results obtained by the study of ergographic curves and the examination of parameters. An excellent idea of this line of investigation will be found in the abstracts of another of Mademoiselle Ioteyko's papers which have already appeared in this Review (Jan. and Feb. 1906, pp. 40 and 128). In the present communication, reference is made to her more recent work on the ergography of sugar and caffeine, and to the alteration in fatigue curves when the arm is rendered anæmic. Her results go to confirm her theory of the peripheral localisation of fatigue.

S. A. K. WILSON.

**PSYCHIATRY.**

**ON IDIOCY.** (Über Idiotie.) By W. WEYGANDT, *Samml. zwangl. (109) Abhandl. aus dem Gebiete der Nerven. u. Geisteskrankheiten*, Bd. vi., 1906.

THIS paper, consisting of 86 pages, is divided into two parts. In Part I. the author gives a brief account of some of the chief clinical and pathological features of the main types of idiocy. He draws attention to the fact that, although primarily due to arrested development, there are frequently added to this secondary pathological changes, resulting in epilepsy, athetosis, chorea, etc. Hence the need for idiots to be under constant medical care. He urges the importance of further research in this field, particularly from a pathological and psychological point of view.

Part II. is devoted to the care of idiots. After referring to the accommodation for this class in the Middle Ages, provided by the monasteries, and thence onwards, the author deals more particularly with the organised attempts of the nineteenth century. He has made a tour of most of the institutions in Germany, Austria, France, and Switzerland, as well as England, and his description of these is very interesting and instructive. He finds that in many instances the accommodation provided for idiots on the Continent is far from satisfactory, not only with regard to the nature of the buildings and general management, but also owing to the absence of any expert medical supervision. In strong contrast to these are the English establishments, all of which are under efficient medical supervision, and the management and general arrangements of which are excellent. Finally, the author discusses the general administrative principles which should guide one in making provision for this—the lowest grade of amentia. The article hardly professes to deal with any new matter, but is an interesting sketch of the chief features of idiocy—especially from the standpoint of administration.

A. F. TREDGOLD.

**ON ACUTE JUVENILE DETERIORATION.** (Ueber acute juvenile (110) Verblödung.) M. FUHRMANN (of Lindenhaus), *Arch. f. Psych.*, Bd. 40, H. 3.

FUHRMANN reports fully the cases of three young male patients, born of alcoholic parents, who developed an acute psychosis resembling symptomatologically an alcoholic psychosis, but passing rapidly into dementia.

The first patient was a man aged 28, who shortly before the onset of the psychosis had shown only a few isolated peculiarities. The psychosis began abruptly with a condition resembling an



epileptic excitement, characterised by extreme anxiety, terrifying hallucinations of sight and hearing, with consequent ideas of persecution. The disease quickly progressed, and on the fourth day there was already a profound disturbance and dissociation of all mental activity; the picture now was more like an alcoholic condition—fantastic and varying hallucinations, dreamy grasp of environment with illusions, incoherence of speech and action, alternation of terror and euphoria, occasional grim humour (Galgenhumor)—but there was a much more profound disorder of the consciousness of the personality than is seen in the similar alcoholic psychosis. The hallucinations became less marked, the acute affect disappeared, and the mood became one of stupid euphoria, and patient in three months was definitely demented.

In the two other patients, aged 25 and 26 respectively, the psychosis began abruptly with hallucinations of a terrifying character, explained by the patients as due to strange influences such as underground telephones, and leading to conditions of marked anxiety; the hallucinations were not only of spoken words, but also of less elaborate nature, *e.g.* flashes of light, shots, twittering of birds. There were frequent variations of mood, and intervals of euphoria in the midst of periods of anxiety and agitation. In the acute phase orientation, grasp of general relations and stream of mentation were unimpaired. The whole picture resembled the acute hallucinatory insanity of the alcoholic; the course, however, was very different. Within three weeks the patients passed into deep stupor and then into dementia, with dull emotional life, lack of initiative, little reaction to the environment, and almost complete mutism.

Fuhrmann refuses to group these cases with the deterioration group of dementia præcox, because the characteristic mannerisms, stereotypies, hypochondriacal ideas, catatonic symptoms, etc., are absent; and also on account of the extremely rapid course of the disease. He therefore describes them as cases of acute juvenile deterioration. With regard to the ætiology, he evidently believes that because the fathers have eaten sour grapes the children's teeth may be set on edge, and suggests that the alcoholic colouring of the clinical picture is due to the excesses of the parents.

C. MACFIE CAMPBELL.

**SYMPTOMATOLOGY OF CATATONIA.** (*Zur Symptomatologie der (111) Katatonie.*) VON LEUPOLDT, *Giessen. Klinik f. psych. u. nerv. Krankheiten*, Bd. 1, H. 1.

DR VON LEUPOLDT describes at considerable length an instructive case of catatonic dementia præcox. The most prominent feature whilst the patient, a young man, was under observation was his

continual and spontaneous naming and enumeration of things in his neighbourhood, particularly those depending on optical impressions. He was well orientated and evidenced great sharpness of perception and clear recollection of past impressions. The naming was almost always a single and rarely a complex process, without any logical or associative connection, and further, without any discoverable dependence on affective disturbance. Elementary or superficial investigation of the power of attention according to Ranschburg's method, revealed no marked defect, but in a more severe memory test (committing to memory and repeating a page of a historical primer) showed a rapidly failing and distracted attention due to the insurgence of optical impressions, with a tendency from complex to simple presentations. The compulsion (*Zwang*) to name objects was not recognised by the patient as foreign, had no affective basis, and was thus not *imperative* in the strict meaning of the term. At the same time the author distinguishes this "naming" from Ziehen's "hyperprosexia" and Wernicke's "hypermetamorphosis." An analogy is next drawn between this phenomenon and that of the continual handling of objects frequently observed in cases of dementia præcox. This latter is illustrated by clinical notes of another case, in which, like the first, there was compulsion without any affective basis or consciousness of the imperative nature of the act. A third case is mentioned, showing that these phenomena may be equally present in the paranoid form of dementia præcox, and the conclusion drawn that the compulsory naming and compulsory touching are consubstantial.

R. CUNYNGHAM BROWN.

**ON THE PSYCHOLOGY OF CONFABULATION.** (*Zur Psychologie der* (112) *Confabulation.*) A. PICK (of Prague), *Neur. Centralbl.*, June 1, 1905, p. 509.

PICK does not discuss here the contents of the fabrications or pseudo-reminiscences which in confabulation serve to fill up gaps of memory; he takes up the question of the psychological basis of confabulation. The existence of a memory gap is not sufficient in itself to explain the phenomenon, for in hysterical or simple traumatic amnesia there may be the memory defect with no attempt to bridge it with fabrications; and while in some cases the symptom is only elicited in answer to an embarrassing question which reveals the defect, in other cases there is a marked tendency to spontaneous confabulation. The explanation of the pseudo-reminiscences on the basis of dream experiences according to Wernicke is inadequate, as one can by suitable questions influence the contents in any direction; Bonhoeffer does not exhaust the phenomenon when he separates the "confabulations of embarrassment" from delirious confabulations, because this only takes into

consideration the cases where the patient is forced by questions to cover the awkward gap in memory, and omits the other cases where there is a spontaneous inclination to confabulate. Spontaneous confabulation is rarer than responsive confabulation, but is very well marked in cases where Korsakow's symptom-complex comes on immediately after trauma. Pick admits as important factors in causing the phenomenon the suggestibility found after trauma, clouding of consciousness, impairment of the critical faculty, and an increased activity of the imagination. Between the cases of mere traumatic amnesia and those with fabrications to supply the gap come cases such as those described by Thorburn after railway accident, which present pseudo-reminiscences, explained by him as due to auto-suggestion in a condition resembling somnambulism. The author quotes a case where a peasant received a severe blow in a quarrel, and during the next ten days had attacks of apprehensiveness in which he forgot the quarrel and gave a false account of the trauma.

According to Pick, in confabulation, images called up by the suggestion of another person or arising spontaneously are projected into the past as memory images; but when we remember an event, we localise it in time and in a certain environment, and so the pseudo-reminiscence is provided with a complete background unconsciously and according to the laws of association and without the direct volition of the patient. C. MACFIE CAMPBELL.

**ON THE CLINICAL SIGNIFICANCE OF CONFABULATION.** (Zur (113) *klinischen Beurtheilung der Confabulation.*) C. NEISSER (of Bunzlau), *Neur. Centralbl.*, Aug. 16, 1905, p. 738.

NEISSER calls attention to the fact that in certain cases of functional psychosis the retrospective falsifications dominate the picture and give the clinical stamp to the case (Paranoia confabulans): such cases were described by Sander under "original paranoia," but are considered by Kraepelin as a mere variety of paranoïdes. Neisser emphasises the fact that in these pseudo-reminiscences there is present neither the clouding of consciousness nor the impairment of the critical faculty which Kraepelin and Pick regard as important factors in the genesis of confabulations. The author regards them not as a secondary phenomenon, but as an independent irritative symptom, and to support his view cites a case where such pseudo-reminiscences developed acutely as an independent episode lasting only a short time.

He refers to the fact that in functional psychosis, especially of a depressive character, a flood of memories may episodically dominate the picture, and this too he would consider an irritative phenomenon. C. MACFIE CAMPBELL.

**ON DEMENTIA PARALYTICA AFTER TRAUMA.** (Ueber (114) *Dementia paralytica nach Unfall*.) By G. REINHOLD (of Crefeld), *Neur. Centralbl.*, July 16, 1905, p. 641.

PATIENT was a man, 40 years of age, with no history of syphilis or of alcoholic excesses, who, after a fall, in which he landed in the sitting position, developed in a few months sluggish pupil reaction, increase of patellar reflexes, unsteady gait. The diagnosis at first was of a functional disorder due to trauma, but the symptoms were progressive and about eighteen months after the accident mental symptoms appeared. Patient showed impaired memory, had little periods of excitement and confusion, fabricated, and had hallucinations. One week before death he had an epileptiform attack. Post-mortem examination disclosed thickening of the pia, atrophy of the convolutions, hydrocephalus internus, granulations of the ventricles, slight atheroma of the aorta.

Neither macroscopical nor microscopical examination showed syphilitic changes in the brain nor other organs.

The author reviews the opinions of various authors on trauma as an etiological factor in general paralysis, and considers that the case recorded was of traumatic non-syphilitic origin.

C. MACFIE CAMPBELL.

**PHYSIOLOGICAL AND PATHOLOGICAL "SLEEP DRUNKEN- (115) NESS."** (*Die physiologische und pathologische Schlaftrunkenheit*.) H. GUDDEN (of Munich), *Arch. f. Psych.*, Bd. 40, H. 3.

THE name "sleep drunkenness" is applied to the state of an individual in whom the transition from the sleeping to the waking state is slow and accompanied by a misinterpretation of the environment and frequently by irresponsible talk and action; the individual is in a half-dream state, the real world is only hazily grasped as in a state of drunkenness. Gudden reports briefly the observations of 18 individuals who presented this phenomenon; he groups the observations in four categories.

Group 1 includes 3 cases of physiological "sleep drunkenness"; this is a quite familiar phenomenon and is illustrated by the first case, who, when wakened one morning after a very short sleep, seized the waker by the throat under the impression that he was a robber, and held him fast until he wakened up completely and grasped the situation.

The occurrence of this phenomenon is favoured by fatigue, too short sleep, uncomfortable position, unfamiliar environment, sudden interruption of sleep.

When the normal individual awakens, grasp of environment

and capacity for action return together ; in "sleep drunkenness" there may be a dissociation. In that case the grasp may return slowly while the individual has no difficulty in moving or talking, or he may have quite clear grasp but be unable to either move or talk for a short time. Two other factors influence this state: the feeling of discomfort associated with premature or disagreeable interruption of sleep, and the strength of the impressions received from the environment before going to sleep. If one fall asleep after only a cursory impression of an unfamiliar environment, orientation on awakening is delayed.

Group 2 includes 2 cases of affective "sleep drunkenness," which are transition cases to pathological examples of the phenomenon. Here the ordinary predisposing factors were accompanied by a certain anxiety due to unfortunate experiences, and the individuals when roused prematurely from sleep, killed another person under the influence of their apprehensiveness before they had grasped the real situation.

Group 3 includes 3 cases which presented the phenomenon in a morbid degree and for which Gudden suggests the name "dream drunkenness" (*Traumtrunkenheit*).

The individuals were of poor heredity, were subject to terrifying dreams, and committed homicide in a condition akin to that of the sleep-walker.

In group 4 are given 9 cases, of which one is from personal observation, under the heading of "alcoholic sleep drunkenness."

In these individuals there was a psychopathic constitution, and the clouding of consciousness was more profound and more prolonged than in the previous groups. The condition was to be distinguished from a pathological condition of intoxication by the suddenness of its occurrence and the quick return of a clear grasp of affairs.

The author's conclusions are as follows :—

1. The most striking sign of "sleep drunkenness" is a delay in the return of grasp of the environment and of capacity for action.

2. The want of definite impressions before going to sleep favours the appearance of the phenomenon.

3. Previous apprehensiveness is a predisposing factor.

4. The feeling of discomfort that accompanies premature awakening influences the actions of the individual in the half-dream state.

5. Pathological "sleep-drunkenness," if not treated with tact, may be prolonged, and in alcoholics may pass over into the pathological excitement of the intoxicated.

C. MACFIE CAMPBELL.

**A CONTRIBUTION TO THE SYMPTOMATOLOGY OF DELIRIUM**(116) **TREMENS.** (*Zur Symptomatologie des Delirium tremens.*)M. REICHARDT (of Würzburg), *Neur. Centralbl.*, June 16, 1905.

THE author has found the following method of use in the examination of patients with alcoholic delirium. He gives the patient a large sheet of white paper and asks him what he sees on it: none of his delirious patients failed to describe a great variety of objects seen; while in cases presenting a similar picture on a non-alcoholic basis, *e.g.* in certain cases of general paralysis, this simple method failed to elicit hallucinations.

The author considers the phenomenon as pathognomonic for the alcoholic delirium, and emphasises the fact that hallucinations can thus be elicited at a time when the patient is clearly oriented, and shows no hallucinations either spontaneously or on pressure of the eyeballs, and in cases of mild and abortive delirium. Its diagnostic importance is considerable in cases not in hospital and which can only be observed for a short time.

The hallucinations are evidently of central origin, because the patients may show normal acuity of vision and no peripheral disorder at the time.

C. MACFIE CAMPBELL.

**PSYCHIATRIC OBSERVATIONS ON A CASE OF MURDER AND**(117) **SUICIDE, ETC.** (*Psychiatrische Untersuchung eines Falles**von Mord und Selbstmord mit Studien über Familiengeschichte und Erblichkeit.*) R. SOMMER, *Klinik f. psych. u. nerv. Krankheiten*, Bd. 1, H. 1, 1906.

SHORTLY before Christmas 1903 a whole family, consisting of a man, his wife, and three children were found dead or dying from severe wounds in a small town in Upper Hesse. The wife and one daughter were already dead, another daughter in a dying condition, and another severely wounded by an axe found in the room. The father, a farmer, had shot himself through the mouth and died two days later. The absence of any apparent motive, and the impossibility of obtaining any information as to the crime beyond external evidence and the statement of the youngest daughter, aged 12, that her father had not done it, and also a blood-stained letter conveying his farewell to his sister-in-law ("Liebe Schwägerin, wenn Ihr meine Frau noch retten könnt, gebt Euch Mühe, letzten Gruss.—S—— X——"), invested the case with considerable psychological interest. Prof. Sommer has been at considerable pains to collect evidence bearing on the crime, and has made an exhaustive inquiry into the personal and family history of the murderer. From the personal history

of the actor, the author was able to establish a morbidly increased susceptibility (*Beeinflussbarkeit*); mental depression following, but out of proportion to, domestic worries; and great motor excitability, hystero-epileptic in nature; with, as exciting or suggesting factors, the proposed slaughter of his pigs, and the smell of blood from some which had been killed on his premises on the previous day. Investigations into the family history, direct and collateral, revealed an extraordinary proportion of neuro-psychopathies, hystero-epileptic in kind, with a marked tendency to emotional disturbance.

R. CUNYNGHAM BROWN.

### TREATMENT.

**HAS FORMIC ACID A MUSCULAR-TONIC ACTION?** (*L'Acide formique a-t-il une action toni-musculaire?*) FLEIG, *Arch. gén. de méd.*, Oct. 31, 1905, p. 2753.

BELIEF in the therapeutic properties of formic acid has oscillated between periods of extreme faith to periods when it has been consigned to utter oblivion. Of late years its virtues have been vaunted more widely than ever. Kowacs was the first within recent years who drew attention to the exciting properties of formic acid upon the motor nervous system, as contrasted with its merely tonic action (*Centralb. f. klin. Med.*, 1885, p. 543).

Garrigue stated that the formates were able to cure such diseases as cancer and tuberculosis ("Maladies microbiennes, guérison de la tuberculose et du cancer"). He stated that these salts, when injected into animals, produced both cerebral and physical activity. Clément, struck by the incessant activity of ants, tried to establish a connection between the presence of formic acid and aptitude for work, and he believed that ants owed their proclivity to work and resistance to fatigue to this acid (*Lyon médical*, 3 août 1903). This author says that formic acid is remarkable for its tonic action, for its power of raising physical and moral force, for its influence on arterio-sclerosis, on senile tremor, and on the other incidents of age, that it preserves youth and retards the onset of old age.

Huchard (*Bull. Acad. méd.*, 14 mars 1905) partially confirmed the opinions of Clément, and drew attention to the value of the formates in cases where one desired to increase the resistance to fatigue in healthy persons, or in such pathological conditions as asthenia, anæmia, influenza, asthenic pneumonia, etc., and drew attention to the diuretic effect of the formates through their action on the muscular fibres of the vessels.

C. Fleig, in an elaborate memoir (*Archiv génér. de méd.*, 31 oct.

1905), criticises the statements of the above authors, and describes a large number of his own experiments. These demonstrate that formate of sodium given in the most varied dose does not modify to any degree the contraction of non-striped muscular fibres, and he is forced to the conclusion that formic acid is not a muscular tonic. Clinical experience shows that the arterial tension undergoes no modification consequent on the exhibition of formates. Fleig could not confirm Clément's statement that the formates had an exciting or reinforcing action on the heart; but if the dose were large, certain toxic effects were noted, as diminution in the amplitude and in the frequency of the heart-beats, as well as a certain degree of arrhythmia. When sodium formate was given to patients suffering from persistent cardiac arrhythmia, negative results were obtained.

Clément and other authors were even more positive in their assertions regarding the effects of formic acid on striated muscular fibres. The former affirmed that this acid diminished, greatly retarded, or even entirely suppressed the sensation of fatigue. Fleig, on the other hand, shows that the idea of it diminishing the sensation of fatigue is illusory.

In order to determine if formic acid acted peripherally either on the nerve fibres or on the muscle fibre itself, Fleig has compared myographic tracings from an animal both before and after the administration of formic acid. These records were obtained by the stimulation of a motor nerve or of a muscle directly. All his experiments showed that it was impossible to determine any stimulating effect either in muscular force or in resistance to fatigue after formate of sodium had been administered.

Many experiments proved that formic acid did not produce muscular stimulation through peripheral action. Nor did myographic tracings produced by direct stimulation of the cord show increased activity after the injection of formates. Neuro-reflex and cutaneous periphoro-reflex contractions were not augmented under the action of these salts. The brain itself did not react more than the cord under the action of formic acid, as judged by the stimulation of certain cortical centres.

Sodium formate was added to the food given to a dog which was made to do work. The observations showed that the salt given in very varying doses neither increased the activity of muscles nor augmented the resistance to fatigue. All these experimental results were very clearly opposed to the assertions of Clément. Fleig goes on to criticise very fully and adversely the statements of this writer as to the benefits conferred by the formates as shown in his ergographic experiments. In conclusion, Fleig states that his observations show that the reputed muscular tonic action of the formates has not been proved. The only stimulating effects which formic acid appeared to produce were a



certain increase in the appetite, though this exhibited itself in a very inconstant manner, and an increased diuresis. In the treatment of certain morbid conditions where asthenia predominated, the employment of formic acid was equally inconclusive.

W. G. AITCHISON-ROBERTSON.

**SOME USEFUL PRINCIPLES IN THE TREATMENT OF**  
**(119) CEREBRO-SPINAL MENINGITIS. WILLIAM BROWNING,**  
*Pediatrics*, Nov. 1905, p. 702.

CEREBRO-SPINAL Meningitis is receiving a good deal of attention at present, especially in the American medical papers. Dr Browning thinks that in most cases definite aid can be obtained from promptly initiated and systematic treatment. He lays special stress upon:—

(1) Light and fresh air. Epidemics occur chiefly in wintry and stormy weather. Patients should be placed in bright, well-ventilated rooms, in sunshine if possible, the eyes being shaded if there is photophobia.

(2) Quiet. The patient must be disturbed as little as possible. Transportation to hospital, though often necessary, does harm. Even removal from one room to another may be followed by retrogression. If the room cannot be kept quite quiet, cotton wool may be placed in the ears. The bed should be comfortable, with a slight incline towards the foot to favour the gravitation of body fluids away from the head. While changes of position to relieve pressure and hypostasis are necessary, they should be as few as possible. During convalescence, too early attempts at rising are very apt to bring on relapses.

(3) Relief of cerebro-spinal congestion. Warmth to the surface is of great importance. Hot-air baths, vapour baths, hot bran bags are useful. Small children may be gently lifted into a bath, but this should not be done to adults. Temporary benefit may result, but the patient suffers for the disturbance in the long run. If the temperature is too high, a cold pack, followed by a warm one, may be used.

Lumbar puncture is of very limited therapeutic value, but often relieves vomiting.

Small blisters along the margin of the occipital and temporal scalp are useful.

Careful nursing is of great importance. Drugs are of very limited value. Stimulants are often required in the later stages.

W. B. DRUMMOND.

**THE DIET IN EPILEPSY.** A. J. ROSANOFF, *Journ. of Nerv. and* (120) *Ment. Dis.*, Dec. 1905, p. 753.

REFERENCE is made in the first place to Merson's observations (*West Riding Asylum Medical Report*, 1875). Twelve epileptic patients were kept for four weeks on a "farinaceous diet," while a similar group were kept on a "nitrogenous diet." The effect of the farinaceous diet was to diminish, that of the nitrogenous diet to increase, the frequency of the convulsions. In a second experiment the first group of patients were given the nitrogenous, the second group the farinaceous diet, with the same effect.

The author holds that there is good ground for the assumption that the various manifestations of epilepsy are probably dependent upon a disorder of nitrogenous metabolism, and upon nothing else, and that in epilepsy there is a hitch in the process of conversion of proteid material into urea, abnormal and often violent activity of the nervous and muscular tissues being necessary for the completion of the conversion. Krainsky has produced convulsions in guinea-pigs by injecting the blood of epileptics. To the periodical accumulation of ammonium carbonate in the blood, the occurrence of convulsions has been attributed. Dr Rosanoff considers that it is not the kind of food, but the absolute quantity of proteid matter, irrespective of its origin, that influences the occurrence of attacks.

Eleven cases of old-standing epilepsy were kept on five different diets for several weeks at a time. (a) Regular diet of the hospital, (b) vegetable diet, (c) diet with insufficient quantity of proteids, (d) diet with excessive amount of proteids, (e) diet with large excess of proteids and very deficient in carbohydrates (the dietetic diet of the hospital). The conclusions arrived at were, that the effect of a mixed diet differs in no way from that of a vegetable diet containing the same quantities of proximate principles, that the quantity of proteids has a decided influence (if it is either above or below the indispensable minimum the severity of the disease is increased); when a diet contains a large excess of proteid and practically no carbohydrates, there is a very great increase in the frequency of the convulsions with an aggravation in the patient's physical and mental condition.

The practical therapeutic deduction from these experiments is that the patient should receive as large amounts of carbohydrates and fats (which are capable of replacing the proteids) as he can assimilate, and the smallest amount of proteids which is compatible with the preservation of the nitrogenous equilibrium.

EDWIN BRAMWELL.

**THE FOOD FACTOR IN THE PAROXYSMAL NEUROSES.**(121) FRANCIS HARE, *Practitioner*, Feb. 1906, p. 179.

THE author argues that the recurrent affections—migraine, asthma, major epilepsy, and acute articular gout—depend primarily upon an accumulation of unoxidised or imperfectly oxidised carbonaceous material in the blood; and that each paroxysm is a conservative measure adopted to disperse such accumulation.

Whatever tends to reduce the carbonaceous income or increase the carbonaceous expenditure of the blood, tends to prevent, alleviate, or disperse; whatever tends to increase the carbonaceous income or decrease the carbonaceous expenditure of the blood, tends to initiate, precipitate, or intensify the paroxysms of migraine, asthma, and epilepsy.

For the restriction of the carbonaceous intake, the following regime is advised: (1) Cutting off sugar and all articles containing it; (2) carefully graduating (by weight) the daily intake of starch-containing foods, so as to attain the minimum consistent with adequate nutrition in each individual case; (3) graduating in a similar manner the intake of fats, if necessary; (4) throwing the onus of nutrition to a considerably greater extent than previously on fish, lean meats, green non-starchy vegetables, and gelatinous soups.

Marked benefit results in most cases, and cure in some cases of migraine and asthma. In epilepsy the results are less satisfactory, but many cases are distinctly benefited and none are rendered worse.

In certain cases of the paroxysmal neuroses, factors other than humoral may be dominant in causation, and therefore worthy of the first consideration in the therapeutic attack.

W. B. DRUMMOND.

**ON THE TREATMENT OF NEURALGIAS BY ALCOHOL INJECTIONS.**(122) TIONS. (Zur Behandlung der Neuralgien durch Alkoholeinspritzungen.) SCHLOESSER (Munich), *Berl. klin. Wochenschr.*, Jan. 15, 1906, p. 82.

THE writer refers to a previous paper in the same journal by Ostwalt (No. 1 for 1906) upon the hypodermic injection of alcohol for neuralgia, and regarding Ostwalt's statement that 90 per cent. of cases yielded excellent results to this treatment, declares that no case (except those of hysterical or diabetic pains) had left his own clinique without being cured. He states that they remained well for six or seven months on an average.

The writer does not describe his method, but promises later an extended publication of his cases.

JOHN D. COMBIE.

**THE INDICATIONS FOR TRANSPLANTATION OF TENDONS.**

(123) (*Die Indikationen zur Sehnenverpflanzung.*) ADOLF LORENZ  
(in Wien), *Wien. med. Woch.*, 1906, p. 118.

PROF. LORENZ inaugurates in this article a reaction against the excessive employment of tendon-transplantation. The object of transplantation is, generally speaking, the equalising of the action of antagonistic muscles when their normal equilibrium has been disturbed. Irrational transplantations, however, often result, not in equilibrium, but in the establishment of a deformity exactly the reverse of the original condition. In pes calcaneo-valgus, for example, the transference of both peronei into the tendo Achillis is apt to result in a paralytic equino-varus, which interferes with progression much more than the original deformity, while one peroneus is quite an inadequate substitute for the paralysed gastrocnemius. In paralytic contractures at the knee it is especially necessary to avoid excessive dynamical changes. Transplantation of the flexors into the quadriceps in genu flexum, or of the quadriceps into the flexors in genu recurvatum, will sooner or later always result in the establishment of the opposite contracture. Genu flexum is far the commoner, and its conversion into a genu recurvatum leaves the patient far more helpless than before; hence transplantation of the flexors into the quadriceps is always inadvisable.

The treatment of conditions such as those mentioned consists, according to Lorenz, in correcting the deformity by a plastic operation, involving, if necessary, division of fasciæ, tendons, or bone, and then in strengthening the weaker muscles by exercises and massage. In most cases nothing more is required, the over-stretched muscles recovering when the tension is thus removed. In a few cases a transplantation of tendons may be necessary, but this should only be performed at a second operation several months after the deformity has been remedied, and when all improvement has ceased. Transplantation should never be practised till paralytic changes have ceased to be progressive, lest the reinforcing muscle should subsequently become involved in the paralysis. Arthrodesis of the ankle-joint the author has never found to be necessary. In spastic conditions, transplantation of the contracted muscles undoubtedly diminishes the spasm, probably by removing the irritation of the tension and not through any alteration in the central innervation or in the direction of nerve currents. A simple tenotomy, however, is as effective.

Prof. Lorenz's assertions refer only to the lower extremity. He admits that in paralysis of the hand transplantation of tendons puts mere correction of deformity entirely into the background.

W. J. STUART.

## Reviews

### **KLINIK FÜR PSYCHISCHE UND NERVÖSE KRANKHEITEN.**

ROBERT SOMMER, Prof. a. d. Univer. Giessen, Bd. 1, H. 1, 1906.  
Halle a. S. : Carl Marhold.

ADVANCE notices of the appearance in serial numbers of clinical studies under the editorship of Prof. Sommer of Giessen, the well-known author of the "Diagnostik der Geisteskrankheiten" and other works, have been for some time before the medical public. In the introduction to this, the first number, Prof. Sommer gives an account of the origin and future scope of this "Klinik."

The extraordinary accretion to medicine in the last few years of clinical and psycho-pathological material, resulting from the rapid development of more exact methods of inquiry, permits to-day of a more accurate diagnosis and much improved treatment in neurological and psychiatric practice. For the past ten years Prof. Sommer has conducted a clinic for nervous and mental diseases, and has, as readers of his works are aware, directed particular attention to, and done much to further, exact methods of clinical investigation in this field. It is, therefore, eminently fitting that this publication should be conducted under his supervision. Under his guidance a school of diagnosis has been formed which has already amassed a large body of clinical material, and with the collaboration of other workers who have been asked to contribute original studies in neuropathology, this material will be published in quarterly numbers, each containing five or six separate articles. Particular consideration is to be devoted to those cases lying on the borderland between nervous and so-called mental diseases, to nervous diseases accompanied by mental symptoms, to those as yet included within the category of functional diseases, hysteria, neurasthenia, and so forth, and their definition and classification more accurately delimited. To the general physician the early stages of the psychoses and psycho-neuroses are of primary importance, and if the aim of Prof. Sommer in this direction is fulfilled and this clinic made to reflect the most modern methods of examination of elementary mental disorders, it cannot fail to be of service to many. In this country particularly, where psychiatric clinics are as yet unfortunately wanting, it should prove most useful to students and to medical officers of asylums, who on account of the statutory precautions which hedge in the entrances to asylums, rarely see cases except in an already advanced, and in many cases incurable condition, and should to some extent supply this lack. Naturally the result of a more exact diagnosis should be improved treatment, and still further the compilation of many

original studies in clinical research must in the end form a valuable addition to works of reference.

The first number, abstracts of some of the articles of which are to be found on the other pages of this issue, will be found to confirm the arguments in favour of its support by all workers in this field.

R. CUNYNGHAM BROWN.

**GEHIRN UND RÜCKENMARK.** Leitfaden für das Studium der Morphologie und des Faserverlaufs. EMIL VILLIGER. Leipzig: W. Engelmann. 1905. Edinburgh: Otto Schulze & Co. Price 9 M.

THE aim of a text-book on the anatomy of the central nervous system may be to give a description of its morphology without regard to its function; or it may treat anatomy as the basis for the comprehension of function and emphasise the facts which are already recognised to be concerned in the production of the symptoms of disease, and which may be of use in clinical study.

This small text-book of 180 pages belongs to the former rather than to the latter class, despite the evident intention of the author to appeal to students of the function of the nervous system in health and disease, and the consequent result is that it cannot be highly praised as a representative of either.

The greater part of it is devoted to the description of the macroscopical appearance of the brain, and this is often excellent. The description and illustration of the complex rhinencephalon is, for example, very complete and lucid, but the rest of the telencephalon is treated so summarily that one previously unacquainted with its anatomy would gain but little from the perusal of these sections. The descriptive anatomy of the spinal cord occupies only half as many pages as have been devoted to the rhinencephalon; surely an unwarranted utilisation of the space at the author's disposal, if his aim were to write a medical anatomy. Similarly the sections which deal with the cerebellum are occupied by a description of the morphology of its gyri, to the exclusion of that of its connections with other parts. This is due to the author's aim to make his book merely a guide to the student who has a brain to dissect; for more detailed information he must seek elsewhere.

Many of the statements in the cortical localisation of function and on the fibre-connections of the different regions of the brain cannot be tacitly accepted. The motor centres do not, in all probability, extend over the post-central gyrus, nor does "the greatest part of the superior frontal convolution" represent the centre from the trunk muscles. The weight of evidence is also at present against the localisation of the auditory word centre in the posterior

end of the first temporal gyrus. Similarly, though all recent work points to the so-called inferior longitudinal bundle being the projection system which carries visual impressions to the calcarine cortex, it is here still described as an association system between the occipital and temporal lobes. Nor is it generally accepted that Helweg's bundle in the upper cervical cord represents the tractus spino-olivaris, and it is doubtful if the fasciculus sulco-marginalis is composed of tecto-spinal fibres alone.

Despite these exceptions, the book can be recommended to those who desire to gain such a knowledge of the structure of the brain as is possible from macroscopical study alone. It is well illustrated and contains numerous diagrams.

GORDON HOLMES.

**THE TEMPERAMENTS.** (*Die Temperamente, ihr Wesen, ihre Bedeutung für das seelische Erleben, und ihre besonderen Gestaltungen.*) Dr EDUARD HIRT (of Munich). Wiesbaden: Bergmann. Pp. 54.

IN this work the author gives a short description of that part of man's psychical life which is evidenced in the varying play of excitation and reaction by his movements, demeanour, mien, and in particular by his changes of manner, the limits of which are fixed by the temperament. The temperament is a life-long characteristic of the personality, which often brings about reactions quite independent of the intellect and character of the individual, more particularly in the extremer states of psychomotor excitement or inhibition to which all are subject. Many manic and depressive conditions appear to be congenitally temperamental, the severer ones constituting cases of manic-depressive insanity.

After some description of the general laws of psychical processes and experiences, the author considers the types of temperament, or special forms under which these laws are manifested. The temperament of the average man-in-the-street is summed up in an inventory of negative qualities. Of the four main types, the Phlegmatic is the nearest to the average—a mere step below the plane of normal psychical energy. Its main characteristic is a sensory blunting, and marked instances grade into undoubted morbid apathy. A sub-class is formed of the Pseudo-Phlegmatics, who have drifted into apathy, often after some attack of over-excitement—they are distinguished by the clear insight they have into their condition, feeling it bitterly; when fully developed, the condition belongs to the class of manic-depressive diseases. Others appear to be mild forms of dementia præcox, the symptoms appearing about the time of the normal termination of mental development.

Contrasted with this type is the Choleric temperament, irascible, but at the same time tenacious of purpose and masterful. This includes the distrustful, suspicious egoists who shade off into paranoid cases, and also the sulky, discontented, bitter people with periods of reproachful sadness, who also form a transition type to the manic-depressive condition.

The third type is the Melancholic, sad, weary, and grave, born to "Weltschmerz." Sub-divisions of this class are—

(a) The self-centred, diffident pessimist.

(b) The conceited melancholic, coming midway between the melancholic and the choleric type, and varying from the man of action to the man of introspective thought.

(c) The man of moods, alternating between moderate excitement and shallow ill-humour—the mood blinding the mind to all real joy and sorrow.

Contrasted with the Melancholic is the Sanguine temperament, full of the *joie de vivre*, but flighty and unstable. This type in an exaggerated form is the slave of momentary impulse, also moody, but the moods are dependent on some evident outward cause.

Many neurasthenics and neurotics fall into this class, and the hysterical temperament may also be considered a sub-class of the Sanguine, its unreliable capriciousness being not hypocritical, but due to some fundamental defectiveness in the will processes.

This classification may or may not be sufficient, individual examples will certainly occur to one that would be found difficult to place in the scheme, but the book is of distinct value as an aid to the study of the temperaments, an application of which, either consciously or unconsciously, must be of the greatest value to one in practice, particularly in dealing with "nervous" or with borderland mental cases.

J. H. HARVEY PIERCE.



# Bibliography

## ANATOMY

- V. BECHTEREW. Über Messung des Gehirnvolums. *Neurol. Centralbl.*, Feb. 1, 1906, S. 98.
- TROLARD. Des radiations du septum lucidum et du trigone. *Rev. Neurol.*, fév. 15, 1906, p. 115.
- KIRSCHMANN. Normale und anormale Farbensysteme. *Arch. f. gesamte Psychol.*, Bd. 6, 1906, p. 397.
- GOLDSCHIEDER. Über die materiellen Veränderungen bei der Assoziationsbildung. *Neurol. Centralbl.*, Feb. 15, 1906, S. 146.
- SCHUMACHER. Über die Nerven des Schwanzes der Säugetiere und des Menschen, mit besond. Berücksicht. des sympathischen Grenzstranges. Hölder, Vienna, 1905, M. 1.20.
- LONDON and PESKER. Ueber die Entwicklung des peripheren Nervensystems bei Säugetieren (weissen Mäusen). *Arch. f. mikroskop. Anat.*, Jan. 1906, p. 303.
- KAPPERS. The Structure of the Teleostean and Selachian Brain. *Journ. Comp. Neurol. and Psychol.*, Jan. 1906, p. 1.
- GEMELLI. Sur la Structure de la region infundibulaire des poissons. *Journ. de l'Anat. et de la Physiol.*, Jan. 1906, p. 77.
- VICTOR BONNEY. A New and Easy Process of Triple Staining for Cytological and Histological Purposes. *Lancet*, Jan. 27, 1906, p. 221.

## PHYSIOLOGY

- MILLS and WEISENBERG. The Localization of the Higher Psychic Functions, with Special Reference to the Prefrontal Lobe. *Journ. of Am. Med. Ass.*, Feb. 3, 1906, p. 337.
- SCHITTENHELM. Untersuchungen über das Lokalisationsvermögen und das stereognostische Erkennen. *Deutsche Zeit. f. klin. Med.*, Feb. 1906, p. 562.
- BACH. Ueber das Verhalten der motorischen Kerngebiete nach Läsion der peripheren Nerven und über die physiologische Bedeutung der Edinger-Westphal'schen Kerne. *Centralbl. f. Nervenheilk. u. Psychiat.*, Feb. 15, 1906, S. 140.
- ALCOCK. The Action of Anæsthetics on the Injury Current of Nerve. *Journ. Physiol.*, Feb. 5, 1906, p. xxvii.
- ELLIOTT. Degenerative Section of the Nerves to the Cat's Bladder. *Journ. Physiol.*, Feb. 5, 1906, p. xxix.
- F. H. SCOTT. On the Metabolism and Action of Nerve Cells. *Brain*, Vol. xxviii., Nos. 111 and 112, 1905, p. 506.
- FÉRÉ. Recherches expérimentales sur l'influence du sel sur le travail. *Rev. de méd.*, Feb. 1906, p. 135.
- BAGLIONI e CURCIO. Ricerche sperimentali sull' azione polare della corrente costante sui centri nervosi. *Zeit. f. allg. Physiol.*, Dec. 1906, p. 613.
- BAGLIONI. Ueber das Sauerstoffbedürfnis des Zentralnervensystems bei Säugetieren. *Zeit. f. allg. Physiol.*, Dec. 1905, p. 415.
- A. B. MACALLUM and M. L. MENTEN. On the Distribution of Chlorides in Nerve Cells and Fibres. *Proc. Royal Soc.*, No. B. 516, Jan. 30, 1906, p. 165.
- OBICI. Di un nuovo metodo per inscrivere grafiche sul cilindro girante. *Riv. di Patol. nerv. e ment.*, Vol. x., f. 12, 1905, p. 545.

## PSYCHOLOGY

- FRANZ. The Time of some Mental Processes with Retardation and Excitement of Insanity. *Am. Journ. of Psychol.*, Jan. 1906, p. 38.
- BROWNE. Psychology of the Simple Arithmetical Processes: a Study of certain Habits of Attention and Association. *Am. Journ. of Psychol.*, Jan. 1906, p. 1.
- CHAMBERLAIN. Acquisition of Written Language by Primitive Peoples. *Am. Journ. of Psychol.*, Jan. 1906, p. 69.
- FERREE. An Experimental Examination of the Phenomena usually attributed to Fluctuations of Attention. *Am. Journ. of Psychol.*, Jan. 1906, p. 81.
- C. H. TOWSE. The Kinæsthetic Element in Endophasia and Auditory Hallucination. *Am. Journ. of Psychol.*, Jan. 1906, p. 127.
- LEHMANN. Beiträge zur Psychodynamik der Gewichtempfindungen. *Arch. f. gesamte Psychol.*, Jan. 1906, p. 425.
- RODENWALDT. Der Einfluss der militärischen Ausbildung auf das geistige Inventur des Soldaten. *Monatsschrift f. Psychiat. u. Neurol.*, Jan. 1906, p. 67.
- GERAUD-BONNET. Transmission de pensée. Rousset, Paris, 1906.
- EDWIN ASH. Some Experiments in Hypnosis. *Lancet*, Jan. 27, 1906, p. 216.
- JOSEPH BELLEI. A Further Contribution to the Study of Mental Fatigue in School Children. *Lancet*, Feb. 3, 1906, p. 287.
- ZBINDEN. Conception psychologique du nervosisme. *Arch. de Psychol.*, T. v., No. 19, 1906, p. 185.
- SCHUYTEN. Sur la validité de l'enseignement intuitif primaire. *Arch. de Psychol.*, T. v., No. 19, 1906, p. 245.
- GERLING. Die Gymnastik des Willens. Möller, Oranienburg, 1906, M. 3.
- KASSOWITZ. Allgemeine Biologie. Bd. 4. Nerven und Seele. Perles, Vienna, 1906, M. 12.
- M. F. WASHBURN. A Comparison of Methods for the Determination of Ideational Type. *Am. Journ. of Psychol.*, Jan. 1906, p. 121.
- M'DOUGALL. On a New Method for the Study of Concurrent Mental Operations and of Mental Fatigue. *Brit. Journ. Psychol.*, Vol. i., Part 4, 1906, p. 435.

## PATHOLOGY

- SCHOLZ und ZINGERLE. Beiträge zur pathologischen Anatomie der Kretinengehirne. *Zeit. f. Heilk.*, Feb. 1906, p. 57.
- BULLARD and SOUTHARD. Cystic Aplasia of the Cerebral Hemispheres in an idiot child. *Journ. of Med. Research*, Jan. 1906, p. 431.
- GUERRINI. Sulla funzione dei muscoli degenerati. *Sperimentale*, Nov.-Dec. 1905, p. 797.
- PELLIZZI. Della eterotopia della sostanza corticale cerebrale. *Ann. de Freniatria*, Dic. 1905, p. 289.
- RABAUD. Pathogénie de la pseudencéphalie (suite et fin). *Nouv. Icon. de la Salpêtrière*, nov.-déc. 1905, p. 675.
- ROUSSY. Un nouveau cas de soi-disant Hétérotopie du Cervelet. (Soc. de Neurol.) *Rev. Neurol.*, jan. 30, 1906, p. 88.
- ROBERTSON, W. FORD. The Pathology of General Paralysis of the Insane. *Rev. Neurol. and Psychiat.*, Feb. 1906, p. 73.
- CHARLES WORKMAN and WALTER K. HUNTER. The Histological Appearances of the Cord and Medulla in a Case of Acute Ascending Paralysis. *Rev. Neurol. and Psychiat.*, Feb. 1906, p. 106.
- BORUTTAU. L'Électropathologie des nerfs amyéliques du poulpe. Blais et Roy, Poitiers, 1906.

## CLINICAL NEUROLOGY AND PSYCHIATRY

## GENERAL—

- CHAVIGNY. Diagnostic des maladies simulées dans les accidents du travail. Baillière et fils, Paris, 1906, 10 fr.
- SOMMER. Klinik für psychische und nervöse Krankheiten. Bd. 1. Marhold, Halle, 1906, M. 3.

- NAEGELI. Nervenleiden und Nervenschmerzen, ihre Behandlung und Heilung durch Handgriffe. Fischer, Jena, 1906, M. 2.  
 HELLPACH. Nervenleben und Weltanschauung ihre Wechselbeziehungen im deutschen Leben von heute. Bergmann, Wiesbaden, 1906, M. 2.  
 ANTON. Über der Wiedersatz der Funktion bei Erkrankungen des Gehirns. *Monatsschrift f. Psychiat. u. Neurol.*, Jan. 1906, p. 1.

**MUSCLES—**

- KLIPPEL et MAURICE VILLARET. Asthénies et atrophies myopathiques (étude synthétique). *Arch. gén. de méd.*, fév. 13, 1906, p. 353.  
 LENOBLE et AUBINEAU. Une variété nouvelle de Myoclonie Congénitale pouvant être héréditaire et familiale à Nystagmus constant. (Soc. de Neurol.) *Rev. Neurol.*, jan. 30, 1906, p. 101.

**PERIPHERAL NERVES—**

- RAYMOND et LEJONNE. Polynévrite consécutive à un Empoisonnement aigu par l'Arsenic. (Soc. de Neurol.) *Rev. Neurol.*, jan. 30, 1906, p. 79.  
 ALBERT KNAPP. Die polyneuritischen Psychosen. Bergmann, Wiesbaden, 1906, M. 4.  
 BRISSAUD et MOUTIER. Un cas de Cellulo-névrite. (Soc. de Neurol.) *Rev. Neurol.*, jan. 30, 1906, p. 91.  
 COMTE. Syndrome Pseudo-bulbaire d'origine Névritique. (Soc. de Neurol.) *Rev. Neurol.*, jan. 30, 1906, p. 94.  
 KLEMPNER. Über Narkoselähmung des N. cruralis und obturatorius. *Neurol. Centralbl.*, Feb. 1, 1906, S. 107.  
 SCHUTZE. Acute aufsteigende (Landry'sche) Paralyse nach Typhus abdominalis mit Ausgang in Heilung. *Berlin. klin. Woch.*, Feb. 12, 1906, p. 201.

**SPINAL CORD—**

- A. M. BARRETT. Spinal cord degeneration in a case of Acromegaly with Tumour of the Pituitary Region. *Am. Journ. of Med. Sci.*, Feb. 1906, p. 246.  
 Tabes.—LONG et CRAMER. Du tabes tardif. *Rev. Neurol.*, fév. 15, 1906, p. 110.  
 D. C. GREENE. A study of the larynx in tabes. *Boston Med. and Surg. Journ.*, Jan. 25, 1906, p. 97.  
 Pott's Disease.—DUPRÉ et CAMUS. Paraplégie pottique par myélomalacie, sans leptoméningite, ni compression, éclosion du signe de Babinski. *Rev. Neurol.*, jan. 15, 1906, p. 1.  
 ROSSI. Sur la pathogénie des altérations médullaires survenant au cours du mal de Pott. *Archiv de Neurol.*, déc. 1905, p. 417.  
 FAURE. Nouvelle méthode de traitement des paraplégies spasmodiques par les Exercices. *Rev. de méd.*, Feb. 1906, p. 164.  
 Hydatids.—A. G. OWEN. Record of five cases of Spinal Hydatid treated by operation. *Inter-Colonial Med. Journ. of Australasia*, Dec. 20, 1905, p. 544.  
 Disseminated Sclerosis.—ANDRÉ-THOMAS et COMTE. Paralysie avec Contracture des quatre membres. Sclérose en plaques vérifiée à l'autopsie. (Soc. de Neurol.) *Rev. Neurol.*, jan. 30, 1906, p. 86.  
 Meningitis.—GAUSSEL. La guérison histologique de la méningite cérébro-spinale. *Rev. Neurol.*, jan. 30, 1906, p. 46.  
 SPRINGTHORPE. Case of Fulminant Coccic Meningitis. *Intercolonial Med. Journ. of Australasia*, Dec. 20, 1905, p. 541.  
 OSBORNE. Treatment of cerebro-spinal meningitis. *New York Med. Journ.*, Feb. 17, 1906, p. 325.  
 Cerebro-Spinal Fluid.—PASHAYAN. Some Notes on the Cerebro-Spinal Fluid. *Med. Record*, Feb. 10, 1906, p. 219.

**BRAIN—**

- Tumour.—KNAPP. The results of operation for the removal of cerebral tumours. *Boston Med. and Surg. Journ.*, Feb. 1, 1906, p. 124.  
 LESZYNSKY. Report of a case of Brain Tumour with Autopsy. *New York Med. Journ.*, Feb. 3, 1906, p. 235.

- BERLINER. Ein Fall von Neubildung des Kleinhirns. *Klinik f. psych. u. nerv. Krankh.*, Bd. 1, H. 1, 1906, S. 65.
- MOCQUIN. Pseudo-tumeur cérébrale par empyème ventriculaire. *Nouv. Icon. de la Salpêtrière*, nov-déc. 1905, p. 651.
- DUPRÉ et CAMUS. Euphorie délirante et Onirisme chez un Phtisique. Double tubercule cortico-méningé frontal symétrique. (Soc. de Neurol.) *Rev. Neurol.*, jan. 30, 1906, p. 90.
- Abscess.**—H. B. STOLL. Abscess of the Brain with a report of five cases. *Am. Journ. of Med. Sci.*, Feb. 1906, p. 223.
- Bulbar Palsy.**—RAYMOND et GUILLAIN. Un cas de Syringobulbie. Syndrome d'Avellis au cours d'une syringomyélie spasmodique. *Rev. Neurol.*, jan. 30, 1906, p. 41.
- Vascular Lesions.**—ERNEST JONES. The Onset of Hemiplegia in Vascular Lesions. *Brain*, Vol. xxviii., Nos. 111 and 112, 1905, p. 527.
- HOGARTH PRINGLE. Some Notes on Hæmorrhage from the Middle Meningeal Artery. *Scot. Med. and Surg. Journ.*, Feb. 1906, p. 97.
- Sclerosis.**—ALFRED W. CAMPBELL. Cerebral Sclerosis. *Brain*, Vol. xxviii., Nos. 111 and 112, 1905, p. 367.
- General Paralysis.**—JOSEPH COLLINS. The Etiology, Prognosis, and Treatment of General Paralysis. Jan. 27, 1906, p. 125.
- KNAUER. Progressive Paralyse. *Münch. med. Woch.*, Feb. 20, 1906, p. 361.
- NÄCKE. Syphilis und Dementia paralytica in Bosnien. *Neurol. Centralbl.*, Feb. 15, 1906, S. 157.
- GIACHETTI. Contributo allo studio della sensibilità nella paralisi generale progressiva. *Riv. di Patol. nerv. e ment.*, Vol. x., f. 12, 1905, p. 550.
- CONOLLY NORMAN. Multiple Lipomata in General Paralysis. *Journ. of Ment. Sci.*, Jan. 1906, p. 62.
- MARANDON de MONTYEL. L'accommodateur dans la paralysie générale. *Journ. de Neurol.*, fév. 5, 1906, p. 41.
- Cerebellum.**—STRÄUSSLER. Zur Kenntnis der angeborenen Kleinhirnatrophie mit degenerativer Hirnstrangerkrankung des Rückenmarks. *Zeit. f. Heilk.*, Feb. 1906, p. 30.
- WHITEHEAD. Three Cases of Cerebellar Abscess. *Journ. of Laryngology, Etc.* Feb. 1906, p. 33.

#### MENTAL DISEASES—

- F. G. CROOKSHANK. Some Notes on the Study of Insanity. *Journ. of Ment. Sci.*, Jan. 1906, p. 49.
- THOMAS DRAPER. A Note on Psychiatric Terminology and Classification. *Journ. of Ment. Sci.*, Jan. 1906, p. 75.
- INGEGNIEROS. Nuova classificazione dei delinquenti fondata sulla psicopatologia. *Ann. di Freniatria*, Dic. 1905, p. 349.
- GATTIE. Lunacy Practice. Simpkin, Marshall, Hamilton, Kent & Co., London, 1906, 2s. 6d.
- PILCZ. Beitrag zur vergleichenden Rassen-Psychiatrie. Deuticke, Vienna, 1906, M. 2.
- J. S. BOLTON. Amentia and Dementia. *Journ. of Ment. Sci.*, Jan. 1906, p. 1.
- RAINSFORD. Necessity for State Interference on behalf of the Imbecile. *Journ. of Ment. Sci.*, Jan. 1906, p. 108.
- NICOLAEVICI. Le délire dans les maladies infectieuses. *Thèse*. Jouve, Paris, 1906.
- SOMMER. Geisteschwäche bei psychogener Neurose. *Klinik f. psych. u. nerv. Krankh.*, Bd. 1, H. 1, 1906, S. 51.
- C. H. TOWN. Negative Aspect of Hallucinations. *Am. Journ. of Psychol.*, Jan. 1906, p. 134.
- LIEBMANN. Epileptische Geistesstörungen. *Deutsche Klinik*, 1905, Lief. 158, p. 540.
- BONHOEFFER. Die alkoholischen Geistesstörungen. *Deutsche Klinik*, Lief. 158, 1906, p. 511.
- WOLLENBURG. Die Melancholie. *Deutsche Klinik*, Lief. 158, 1905, p. 493.
- PFEIFFER. Ueber das Krankheitsbild der "zirkumskripten Autopsychose auf Grund einer überwartigen Idee." *Monatsschrift f. Psychiat. u. Neurol.*, Jan. 1906, p. 49.

- PICK. Ueber einer weiteren Symptomenkomplex im Rahmen der Dementia senilis, bedingt durch umschriebene stärkere Hirnatrophie. *Monatsschrift f. Psychiat. u. Neurol.*, Feb. 1906, p. 97.
- DÉ BUCK et DEROUBAIX. Considérations anatomo-psychologiques sur la démence précoce. *Journ. de Neurol.*, jan. 20, 1906, p. 27.
- KLIPPEL et LHERMITTE. Lésions de la Moëlle dans la Démence Précoce. (Soc. de Neurol.) *Rev. Neurol.*, jan. 30, 1906, p. 93.
- TIRELLI. Perizia medico-legale sulle condizioni mentali di Rosa Bonetti. *Ann. di Freniatria*, Dic. 1906, p. 320.
- SOMMER. Psychiatrische Untersuchung eines Falles von Mord und Selbstmord mit Studien über Familiengeschichte und Erblichkeit. *Klinik f. psych. u. nerv. Krankh.*, Bd. 1, H. 1, 1906, S. 6.
- GEORGE GREENE. Incidence of Tuberculosis in Asylums. *Journ. of Ment. Sci.*, Jan. 1906, p. 92.
- ROGERS. Should the Tuberculous Insane in Hospital be Segregated. *Boston Med. and Surg. Journ.*, Jan. 18, 1906, p. 62.

#### GENERAL AND FUNCTIONAL DISEASES—

- TILING. Clemens Neisser: Individualität und Psychose. *Centralbl. f. Nervenheilk. u. Psychiat.*, Feb. 1, 1906, S. 91.
- EDWARD COWLES. The Problem of Psychiatry in the Functional Psychoses. *Boston Med. and Surg. Journ.*, Feb. 1, 8, 15, 1906.
- Epilepsy.**—SCHMITZ. Aufklärung für Epileptiker. Hiller, Hanover, 1905, M.—50.
- BRA. Recherches neurobiologiques sur l'Epilepsie. *Archiv de Neurol.*, déc. 1905, p. 469.
- ONUF and LOGRASSO. Researches on the Blood of Epileptics. *Am. Journ. of the Med. Sci.*, Feb. 1906, p. 269.
- HOUZEL. De la Saignée dans le mal épileptique. *Presse Médicale*, Jan. 31, 1906, p. 67.
- PLAVEC. Kleine motorische Epilepsie. *Neurol. Centralbl.*, Feb. 1, 1906, S. 111.
- LÉON TIXIER. Rapport des états anxieux et des états épileptiques. *Thèse de Paris*, 1905.
- CHOTZEN. Mischzustände bei Epilepsie und Alkoholismus. *Centralbl. f. Nervenheilk. u. Psychiat.*, Feb. 15, 1906, S. 129.
- RASCH. Ueber die Anwendung von Geheimmitteln bei Epilepsie (Fallsucht). *Buchh. der Anstalt Bethel*, 1906, M.—25.
- ALAN M'DOUGALL. The David Lewis Manchester Epileptic Colony. *Journ. of Ment. Sci.*, Jan. 1906, p. 84.
- Chorea.**—ANDRÉ BRUEL. Traitement des Chorées et des Tics de l'Enfance. Steinheil, Paris, 1906.
- J. SPENCER SHEILL. "Chorea Gravidarum." *Practitioner*, Feb. 1906, p. 192.
- LANGE. Über chronische progressive Chorea (Huntington) in jugendlicher Alter. *Berlin. klin. Woch.*, Feb. 5, 1906, p. 153.
- Hysteria.**—INGEGINEROS. Les prétendus symptômes de l'hémiplégie historique. *Presse méd.*, fév. 17, 1906, p. 105.
- ETIENNE. Ulcère utéro-vaginal phagédénique et gangrène cutanée de nature hystérique. *Rev. Neurol.*, jan. 30, 1906, p. 52.
- DELACROIX et SOLAGER. Amnésie rétro-antérograde générale et presque totale; délire; anesthésie considérable des diverses sensibilités chez une hystérique. *Rev. Neurol.*, jan. 15, 1906, p. 6.
- Neurasthenia.**—DRENKHAHN. Die Nervosität in früheren Jahrhunderten und in unserer Zeit. Seitz & Schauer, München, 1906, M. 1.
- DETERMANN. Über die Nervosität der Jetztzeit und ihre Bekämpfung. Speyer & Kaerner, Freiburg, 1906, M.—90.
- PETIT. Quelques considérations sur le traitement électrique de la neurasthénie. *Bull. de la Soc. Française d'Electrothér.*, jan. 1906, p. 13.
- LÉVY. La cure définitive de la neurasthénie par la rééducation. *Arch. gén. de méd.*, fév. 6, 1906, p. 321.
- FRANCIS HARE. The Food Factor in the Paroxysmal Neuroses. *Practitioner*, Feb. 1906, p. 179.
- Myasthenia Gravis.**—FARQUHAR BUZZARD. The Clinical History and Post-mortem Examination of Five Cases of Myasthenia Gravis. *Brain*, Vol. xxviii, Nos. 111 and 112, 1905, p. 438.

- BOLDT. Ueber einen Fall von myasthenischer Paralyse. *Monatsschr. f. Psychiat. u. Neurol.*, Jan. 1906, p. 39.
- CURSCHMANN u. HEDINGER. Über Myasthenie bei sexuellen Infantilismus nebst untersuchungen über die myasthenische Reaktion. *Deutsche Zeit. f. klin. Med.*, Feb. 1906, p. 578.
- Exophthalmic Goitre.—BEEBE. Preparation of a Serum for Exophthalmic Goitre. *Journ. of Am. Med. Ass.*, Feb. 17, 1906, p. 484.
- ROGERS. Treatment of Exophthalmic Goitre by a Specific Serum. *Journ. of Am. Med. Ass.*, Feb. 17, 1906, p. 487.

## SPECIAL SENSES AND CRANIAL NERVES—

- M'DOUGALL. The Illusion of the "Fluttering Heart" and the Visual Functions of the Rods of the Retina. *Brit. Journ. Psychol.*, Vol. i., Part 4, 1905, p. 428.
- CHAILLOUS et PAGNIEZ. Ophthalmoplégie externe bilatérale congénitale et héréditaire. *Nouv. Icon. de la Salpêtrière*, nov.-déc. 1905, p. 666.
- DREYFUS. Ueber traumatische Pupillenstarre. *Munch. med. Woch.*, Feb. 20, 1906, p. 355.
- ALBRAND und SCHRÖDER. Das Verhalten der Pupille im Tode. Marhold, Halle, 1905, M. 5.
- ADAM. Ein Fall von Abduzenslähmung nach Lumbalanästhesierung. *Munch. med. Woch.*, Feb. 20, 1906, p. 360.
- BLEGVAD. Bemerkungen über Rinne's Versuch sowie über die Bestimmung der Perzeptionszeit von Stimmgabeln. *Nord. med. Ark.*, Afd. 1, H. 2, 1905.
- WITTMACK. Über experimentelle degenerative Neuritis des Hörnerven. *Zeit. f. Ohrenheilk.*, Feb. 1906, p. 161.
- RANJARD. Le vertige auriculaire. *Thèse. Jouve, Paris*, 1905.
- VON LEUPOLDT. Nachweis der Simulation von Taubstummheit durch Schreckwirkung auf akustische Reize. *Klinik f. psych. u. nerv. Krankh.*, Bd. 1, H. 1, 1906, S. 28.
- HUET et LEJONNE. Paralysie faciale et hémistrophie linguale droites ayant vraisemblablement comme origine une polio-encéphalite inférieure aiguë ancienne. *Rev. Neurol.*, fév. 15, 1906, p. 105.
- BALINT. Ein Fall von Fractura basos cranii mit seltener Nervenlähmung. Beiträge zur Physiologie des 9., 10., und 11. Gehirnnerven. *Neurol. Centralbl.*, Feb. 1, 1906, S. 99.

## MISCELLANEOUS SYMPTOMS—

- STEINERT. Neue Beiträge zur Lehre von der Muskelatrophie bei supranuclearen Lähmungen besonders bei der cerebralen Hemiplegie. *Deutsche Zeit. f. klin. Med.*, Feb. 1906, p. 445.
- NOICA et AVRAMESCU. Sur la perte du Sens Stéréognostique à topographie radicaire dans quatre cas de Tabes. (Soc. de Neurol.) *Rev. Neurol.*, jan. 30, 1906, p. 99.
- FREDERICK BATTEN. Ataxia in Childhood. *Brain*, Vol. xxviii, Nos. 111 and 112, 1905, p. 484.
- KRAMER. Die kortikale Tastlähmung. *Monatsschrift f. Psychiat. u. Neurol.*, Feb. 1906, p. 129.
- VON LEUPOLDT. Zur Symptomatologie der Katatonie. *Klinik f. psych. u. nerv. Krankh.*, Bd. 1, H. 1, 1906, S. 39.
- HUET. Réaction de dégénérescence dans le releveur de la paupière. *Ann. d'Électrobiol.*, jan. 1906, p. 8.
- SIMONINI. Exostoses ostéogéniques de développement. *Nouv. Icon. de la Salpêtrière*, nov.-déc. 1905, p. 635.
- LAUNOIS et TRÉMOLIERES. Exostoses multiples Contribution à l'étude des dystrophies du cartilage de conjugaison. *Nouv. Icon. de la Salpêtrière*, nov.-déc. 1905, p. 621.
- RUDLER et RONDOT. Scapulae alatae physiologiques. *Nouv. Icon. de la Salpêtrière*, nov.-déc. 1905, p. 667.
- AUGUST HOCH. A Study of Some Cases of Delirium produced by Drugs. *Rev. Neurol. and Psychiat.*, Feb. 1906, p. 83.
- BERNHARDT. Die Betriebsanfälle der Telephonistinnen. Hirschwald, Berlin, 1906, M. 1.50.
- BYROM BRAMWELL. A Series of Lectures on Aphasia. Lecture II. *Lancet*, Feb. 10, 1906, p. 351.

- DEBRAY. Aphasie sensorielle avec hémianopsie latérale homonyme droite. *Journ. de Neurol.*, jan. 20, 1906, p. 21.
- SYDNEY J. COLE. On Some Relations between Aphasia and Mental Disease. *Journ. of Ment. Sci.*, Jan. 1906, p. 28.
- HENNEBERG. Ueber unvollständige reine Worttaubheit. *Monatsschrift f. Psychiat u. Neurol.*, Jan. 1906, p. 17. (*Schluss folgt.*)
- HENNEBERG. Ueber unvollständige reine Worttaubheit. *Monatsschrift f. Psychiat u. Neurol.*, Feb. 1906, p. 159.

#### TREATMENT\*—

- OSTWALT. Des injections alcooliques au nerf des trous de la base du crâne dans la névralgie faciale rebelle. *La Presse méd.*, fév. 17 et 24, 1906.
- JORDAN LLOYD. On Facial Neuralgia and its Curative Treatment by Excision of the Gasserian Ganglion. *Birmingham Med. Rev.*, Jan. 1906, p. 16. (*To be continued.*)
- HARVEY CUSHING. On Preservation of the Nerve Supply to the Brow, in the Operative Approach to the Gasserian Ganglion. *Annals of Surgery*, Jan. 1906, p. 1.
- MORTON PRINCE. Head Injuries. *Boston Med. and Surg. Journ.*, Feb. 15, 1906, p. 182.
- SACHS. Serious Head Injuries and the Indications for Operative Treatment. *Boston Med. and Surg. Journ.*, Feb. 15, 1906.
- W. N. BULLARD. Indications for Operation in Head Injuries. Feb. 15, 1906, p. 184.

\* A number of references to papers on Treatment are included in the Bibliography under the Individual Diseases.

# Review of Neurology and Psychiatry

---

## Original Articles

### ON TETANOID CHOREA AND ITS ASSOCIATION WITH CIRRHOSIS OF THE LIVER.

By SIR W. R. GOWERS, M.D., F.R.S.

A SHORT outline of the first of the two following cases was given in my "Manual Dis. Nerv. System," vol. ii., 1888, p. 656, under the designation "Tetanoid Chorea," and was quoted by Dr Ormerod in an article (*St Barts. Hosp. Rep.* 1890) in which he described a case somewhat similar, with cirrhosis of the liver, a condition present in both the following cases. They were under observation many years ago, and their record has been waiting for other facts that might elucidate their mystery, but waiting in vain. They are now presented in the hope they may direct further attention to these strange forms of disease.

In the first case the symptoms began in apparent health, and consisted in changing spasm of a tetanoid character, ultimately becoming fixed in the extremities, attended with variable pyrexia and emaciation. It was steadily progressive in course and ended in death in six months. A careful examination failed to disclose any organic disease in the nerve centres. Not the least remarkable feature is the occurrence of analogous affections in other members of the family, and their association in some, with cirrhosis of the liver.<sup>1</sup>

<sup>1</sup> The notes of the case were taken by Dr T. Wilson, Resident Medical Officer, now Obstetric Physician to the General Hospital, Birmingham.



The patient was a boy, Sydney M., aged 10, who was admitted to the National Hospital for the Paralysed and Epileptic on October 5th, 1886. His father's brother is said to have suffered from a similar affection at the age of 16, and to have recovered after an illness lasting 12 months; a sister of his father also had "St Vitus' Dance" at the age of 16, which lasted 3 months; two children of another sister also apparently suffered from some form of chorea and recovered. The patient was the eighth of 16 children, of whom 11 were dead, one from consumption, the others, it was said, from bronchitis; his eldest brother<sup>1</sup> was in the National Hospital six or seven years before, and died from some form of paralysis at the age of 15. The patient had never suffered from rheumatism. His symptoms began three months before admission, without any exciting cause. Clumsiness with his knife and fork first attracted attention, and the awkwardness in moving the hands gradually increased; spontaneous movement developed and affected the legs as well as the arms.

On admission he was found well nourished. His heart was normal. Constant, slowly changing movements in the limbs and arms at once attracted attention. A contraction of the zygomatic muscles caused a continuous smile, now greater on one side, now on the other. The mouth was usually wide open and the tongue retracted, but sometimes, by an effort, he could slowly protrude it. The open mouth was due to spasm in the depressors of the lower jaw. If told to shut his mouth he pressed the lower jaw up with his hand beneath the chin, and after a moment or two the spasm seemed to give way and he closed the mouth easily, but in about a minute the spasm came on again and lowered the jaw. The strong retraction of the tongue interfered much with swallowing, because the tongue was pressed up against the hard palate; liquids taken into the mouth ran out again, unless the jaw were raised up; as the jaw began to descend again, he was able to flatten his tongue sufficiently to allow the liquid to pass into his throat, but the tongue immediately resumed its rigid position. This process had to be repeated with each mouthful.

<sup>1</sup> He was said, in the history of this patient, to have died in the Hospital. All efforts to trace the case in the Hospital Records have failed. In the history of the sister's case, to be given presently, he is said to have died at home, some months after his discharge from the Hospital.

He seldom tried to speak, but occasionally managed to utter a sentence which could be understood, especially in the morning. The spasm was always less after sleep, and worse as the day went on. He almost constantly made a low whining sound.

The movements of the eyes were normal, but at times the balls were rolled upwards. There was much spasm in the neck muscles, especially at the back, so that the head was almost constantly bent backwards; there was also strong spasm in the sterno-mastoids. Occasionally, however, when he sat up in bed, his back and neck were arched forwards so that his head was between his knees.

Both arms presented slowly changing tonic spasm, greater in the left. The forearms were usually pronated; the fingers half flexed at all joints, and the thumb also flexed. From time to time the spasm increased, and then the elbows became strongly extended, the arms adducted at the shoulder. Voluntary movement was interfered with by the spasm to a less extent than might be anticipated; he could take hold of any object with a little difficulty. At times the spasm changed so that the fingers were spasmodically extended; occasionally they were spread out and moved irregularly in a manner resembling athetosis, now and then more quickly, but the constant tonic spasm prevented any actual resemblance to ordinary chorea. It was always increased by an attempt at voluntary movement, even when this could be effected.

In the legs there was similar spasm, also a little greater on the left side. The left foot was in constant strong extension at the ankle, and inverted; the spasm could not be completely overcome by passive force. It varied less than in the arms, but occasionally passed off entirely for a short time. The right foot presented very little spasm, but at times the leg was extended at the hip and knee by spasm which came on gradually and slowly passed away. At the hips the spasm occasionally changed to flexion, and the leg, still extended, became flexed on the trunk at an angle of about 60°. The boy was able to walk; the spasm in the left foot generally prevented the left heel from reaching the ground, but now and then relaxation of the calf muscles permitted him to walk naturally. The abdomen was generally retracted by spasm in the abdominal muscles, distinctly greater on the left side.

When the body was spasmodically bent forwards the spine presented one long curve, with a slight lateral deviation in consequence of the stronger action of the muscles on the left side. The knee-jerk could be obtained on each side, but was slight, apparently from the interference of the spasm. There was no foot clonus. The plantar reflex was slight, the cremasteric active; no abdominal reflex could be elicited. Mechanical irritability of the nerves was repeatedly searched for in the limbs, but could not be found. Sensation was everywhere normal. He complained of some pain in the dorsum of the left foot. His optic discs were normal. His mind seemed unaffected. When the spasm in the tongue prevented him speaking he would write down the word he wanted to say, and evidently understood everything that was said to him. His urine was normal, and so, at first, was his temperature. When he was asleep the spasm passed away entirely, except in the calf muscles of the left leg; the mouth was closed.

During the first few days after admission a distinct improvement occurred, but after the first week he became rapidly worse. His temperature rose to  $100^{\circ}$  and  $101^{\circ}$ ; the spasm had the same character but was greater, and became as severe on the right side as on the left. He ceased to speak intelligibly and became drowsy, and began to pass his urine and stools into the bed.

On October 16th, attacks of spasmodic difficulty of breathing came on. When lying moderately quiet, with the usual spasm of the arms and hands, this would suddenly increase; the mouth opened so widely that the jaw seemed subluxated, and went back with a snap when the mouth was closed. As the jaw descended, the breathing became quicker, and it was seen that the tongue seemed to be drawn up almost into the throat, so as to impede the breathing, until relieved by drawing the tongue forward and forcibly closing the jaw. During the attack the face was flushed but not livid. Such an attack, lasting half a minute, would recur every four or five minutes. His temperature rose to  $101.6^{\circ}$  and his pulse to 168. The attacks ceased on the application of a spinal ice bag. Next day he was much quieter, the pulse fell, but the temperature rose to  $102.6^{\circ}$ ; in the evening the spasms became very violent.

For the next fortnight the condition continued nearly the same,

PLATE 16.





in spite of varied treatment. The temperature continued between  $100^{\circ}$  and  $102^{\circ}$ , and he rapidly lost flesh. Severe paroxysms occurred, in which his respirations were 60 to 80 a minute. The general spasm continued the same in general character, but that in the hands became very uniform. Both were generally strongly flexed at the wrist, the fingers semi-flexed but not forced into the palm, the thumb adducted, the forearm supinated to its full degree so that the back of the hand was downwards. The face presented little spasm, except during the paroxysmal increase, when his features were distorted. These attacks did not seem to involve the legs, but the extreme extension of the feet continued. The spasm no longer ceased during sleep, but continued much as when he was awake; it seemed to cause little suffering.

A month after admission, some improvement occurred; he became able to speak a little. The temperature was generally about  $100^{\circ}$ . Emaciation continued; the muscles wasted, but presented no change of electrical or mechanical excitability. In the middle of November there was again an increase in the spasm. The feet were strongly extended at the ankles and the toes were strongly flexed. The spasm of the trunk was still flexor; the body bent forwards, and the thighs flexed at the hip, raising the legs off the bed, so that the patient seemed balanced on the gluteal region. But after two weeks the spasm again lessened, and such flexion of the trunk became rare, but the plantar flexion of the feet and toes increased, so that the sole became arched. The spasm in the arms continued, but the flexion of the left wrist became less than that of the right. During November the temperature varied, sometimes normal for a day or two, then rising to  $102^{\circ}$ ,  $103^{\circ}$ , or  $104^{\circ}$ . The wasting steadily increased, so that the child was reduced "almost to a skeleton," although a fair amount of nourishment was taken. During the first fortnight of December the spasm was definitely less and the temperature was lower, varying from a little below to a little above normal; occasionally he would talk a little. On December 17th his cheeks and supramaxillary regions were found to be swollen and crepitated on pressure, evidently from air in the cellular tissue. This condition spread down the left side of the neck to the left axilla and left side of the chest, but rapidly lessened in the next few days. The evening temperature was generally  $100^{\circ}$  or  $101^{\circ}$ . The boy

became rapidly weaker, ceased to swallow, mucus accumulated in the chest. On December 22nd the temperature rose to  $104^{\circ}$ , and he died, eleven weeks after admission, and about six months after the commencement of the symptoms. Throughout, his heart presented no murmur and the urine no albumen. The position of the legs and arms produced by the spasm during life continued after death.

A careful post-mortem examination was made, and a thorough naked-eye examination of the brain, spinal cord, and membranes revealed nothing abnormal. The heart was healthy. The cellular tissue of the anterior mediastinum contained air, which extended in front of the trachea, and down to the sub-pleural tissue of the left lung near its root, whence it had evidently escaped. The liver was noted to be "firm, hard, lobular, light in colour, not greasy, and not staining with iodine." (It was evidently cirrhotic, although the significance of this was not realised at the time.) There had been no jaundice.

Portions of the cortex of the brain, of the spinal cord, peripheral nerves and muscles, were hardened and examined microscopically by Dr Wilson, but no deviation from the normal could be discovered.

Two years later, August 29th, 1888, the boy's sister, Charlotte M., aged 15, was admitted, because for nine months she had been restless and lethargic, with some thickness of speech and tendency for saliva to flow from the mouth. At the onset the catamenia, established for eighteen months, had ceased. She was a heavy-looking girl, with mouth generally open and lower lip hanging down, easily excited to laughter. The tongue was rather large, and when protruded had a slight tremulous movement. There was nothing abnormal in the throat except some congestion. She was able to walk well, the knee-jerks were normal; there was no foot clonus, the optic discs were normal, and no other symptoms were found. At the end of six weeks she was sent to the Country Branch. But six weeks later, "choreic movements" commenced in the right leg, and extended to the other leg and to both arms in a fortnight. She was readmitted on January 2nd, 1889. The movements increased during the month before admission, and for a few days the legs had been drawn up, flexed at the hip and knee, while her arms were extended and

raised above her head. Lying thus, there was constant regular movement of the feet, the heels resting on the bed and the toes sharply depressed and then raised; at the knees and hips there were also slight flexor and extensor movements, moving the heels up and down the bed for an inch or two. Similar rhythmical movements occurred in the arms at the elbows. The trunk muscles and those of the neck and head were free, and the face was still. The tongue now presented no tremor or spasm, and could be voluntarily protruded.

The movements varied in degree, and could be occasionally stopped for a few seconds by voluntary effort.<sup>1</sup> There was no tenderness of the muscles or wasting. Passive movements caused some pain, especially attempts to extend the legs. The knee-jerks could not be obtained (perhaps from the spasm); there was no foot clonus.

No derangement could be found in any cranial nerve, pupils, eye movements, or optic discs. The heart was healthy. The urine contained  $\frac{1}{4}$ th albumen (casts are not mentioned). The temperature was raised, and during the first three weeks it frequently reached in the evening  $103^{\circ}$  or  $104^{\circ}$ . She steadily lost flesh and became thinner and more feeble. The movements continued, varying much in degree. At times the forearms were brought in front of the chest, and moved rapidly in flexion and extension.

The only medicine which had a marked effect on the movements was the hydrobromate of hyoscin, but toxic effects prevented its continuance. On the evening of January 23rd her temperature was  $106.2^{\circ}$  (verified by several thermometers), reduced to  $100^{\circ}$  by cold sponging. There was no delirium or headache, the pulse was 180 and respirations 68, although no morbid sign was presented by the lungs. The patient became much more excited, and, apparently in consequence of this, the movements became less violent. The heart sounds continued normal, but bronchitic râles appeared. The albuminuria continued. Twice again the temperature rose to  $106^{\circ}$ . Evacuations were passed into the bed. She died on January 30th, the temperature rising just before death to  $108.4^{\circ}$ .

Post-mortem examination revealed no morbid appearance in

<sup>1</sup> It is perhaps worth while to point out how readily and reasonably the symptoms might have been regarded as "functional," *i.e.* hysterical.



the membranes of the brain; no sign of tubercle could be discerned in them. The white substance of the hemispheres was studded with minute "pits" the size of a pin's head (the significance of which is probably small). No other morbid state could be discovered in the brain. The spinal dura mater was, in places, adherent to the bone, but was not thickened, and presented no other morbid appearance; in the lower dorsal region there was a small extravasation outside the dura mater, but there was no morbid appearance on its inner surface. The spinal cord and sections of this in all parts appeared perfectly normal to the naked eye. The heart was healthy. No sign of tubercle could be found anywhere. In the lungs there was very slight hypostatic congestion.

The liver, of normal size, presented the typical appearance of cirrhosis. Strands of connective tissue enclosed yellow lobules of various sizes rising above the level of the section. The tint was found to be due to fatty and granular degeneration of the cells. The condition of the liver led to careful inquiry regarding alcohol, but it was found she had never taken it. There is no record of a microscopical examination.

The case described by Dr Ormerod was that of a boy of 10, whose symptoms ran their course to death in four months. They bore considerable resemblance to those of Sydney M. described above. There was similar tonic spasm, but apparently it did not present the constantly changing character (which was so marked a feature and suggested the resemblance to chorea). The flexion of the fingers was less strong, and attempts to extend the contracted muscles caused clonic contraction. Towards the last the flexor contraction of the legs was such that the knees were beside the chin, a form of spasm which was only transient in the first case. There was the same irregular pyrexia, less high. After death a small streak of softening was found in the outer part of each lenticular nucleus, presenting only leucocytal infiltration. Two minute spots of softening,  $\frac{1}{8}$  inch in diameter, were found in the pons. The liver was in a state of extreme cirrhosis, fibrous tracts enclosing lobules prominent on the surface, and many softened by degeneration. As far as could be ascertained, there was no reason to think that alcohol had been given to the boy.

Dr Ormerod quotes from Homén (*Neur. Centralbl.*, 1890) a group of three cases in one family, two fatal, in which nervous symptoms with contractures were associated with cirrhosis of the liver. They began at 12, 20, and 26, and were far more chronic in course than those above described. In the two fatal cases, death occurred only after six and seven years. Extensive changes were found in the nervous system—adhesions of the membranes, thickening of the skull, dura mater, and pia mater, and extensive softening of both lenticular nuclei. Homén reasonably assumes inherited syphilis to be the cause of the morbid state and its manifestations.

In the two cases I have recorded no symptom of inherited syphilis was noted. The number of deaths in early life may have been due, more probably, to a tubercular tendency, since they are said to have been caused by "bronchitis," and one death was from recognised consumption. The probability of inherited syphilis as the cause of the malady of the nervous system, rests entirely on the significance of the hepatic cirrhosis, whether or not syphilis is the only cause of the juvenile non-alcoholic form. Even the assumption of this causation leaves the direct pathology as mysterious as before. It is inconceivable, in the first case, and most improbable in the second, that any coarse morbid process could have existed and have escaped the careful examination to which the nerve-centres were subjected. The facts seem compatible only with a blood state as the cause of the symptoms. The occurrence of benign chorea in distant relatives, though possibly accidental, seems of similar significance.

But if a blood-state caused the symptoms, the question arises: Were this and the cirrhosis of the liver the common effects of one cause, or can the blood-state have been the effect of the hepatic disease? Anomalous as the last assumption may seem, it cannot be hastily dismissed. In connection with the difference in the character of the symptoms, there is also the association of the higher fever in the second case with a greater degree of disease of the liver. Toxic blood-states may be complex in causation; one derangement of the chemical processes of the system may induce others, the effects of which co-operate with the first.

I have been unable to find that any other examples of

similar disease or a similar association have been recorded during the last fifteen years, but references to any that have escaped my notice will no doubt be welcomed by the Editors of this Review. I hope that the belated publication of these cases may induce the careful study during life, as well as after death, of any that may be hereafter met with. The suspicions which similar nerve symptoms should arouse, may lead to the discovery of conclusive facts, not only after death, but during life.

---

## **THE PATHOLOGY OF GENERAL PARALYSIS OF THE INSANE**

By W. FORD ROBERTSON, M.D.,  
Pathologist to the Scottish Asylums.

(The Morison Lectures for 1906.)

### **LECTURE III.**

Delivered on 29th January 1906.

GENERAL paralysis of the insane, and the obviously allied disease tabes dorsalis, have in recent years probably given rise to more discussion than any other morbid conditions that specially manifest themselves in nervous disorders. There are at least three special reasons for which it may be said that it is natural that this should have been so. In the first place, general paralysis and tabes dorsalis, both on account of their frequency and of their gravity, are among the most important of all the nervous diseases that the practitioner is called upon to treat; in the second place, it may safely be said, even without risk of contradiction from the most extreme advocate of the syphilitic theory of the origin of these maladies, that there is very much regarding them that has hitherto remained obscure; and, in the third place, general paralysis and tabes dorsalis still rank among the opprobria of medicine, for all efforts to combat them with success have hitherto proved unavailing.

In discussing the problem of the etiology and pathogenesis of these two diseases in the light of the investigations detailed in the two preceding lectures, I wish, as far as possible, to avoid

a controversial attitude. My purpose is simply to endeavour to show that a new and solid edifice can be constructed out of the facts elicited by my colleagues and myself. A passing critical reference to the syphilitic hypothesis is, however, unavoidable. It is the only view of the etiology of general paralysis and tabes that really conflicts with the one I am going to maintain. There are many who think it is already thoroughly established, and if this is really so there is no room for any rival explanation.

As remarked in the first lecture, there can be observed in the recent literature of general paralysis and tabes dorsalis an increasing dissatisfaction with the syphilitic hypothesis, and a steadily growing conviction that the essential etiological factor has yet to be discovered. The most able discussions of the question that I am acquainted with are those that are to be found in the recently published works upon insanity by Professor Bianchi of Naples<sup>1</sup> and Professor Tanzi<sup>2</sup> of Florence. Professor Bianchi recognises that syphilis is one of the causes of general paralysis, but he does not admit that it is the specific etiological factor. He attaches almost equal importance to alcoholism, and enumerates also many other predisposing factors. He has observed several cases in which general paralysis developed during the secondary stage of syphilis, and this fact, he maintains, is inconsistent with the view that general paralysis is a tertiary or quaternary syphilitic infection. In common with several other observers, he has seen cases in which general paralysis has preceded infection by syphilis. Among numerous other facts likewise tending to weaken the syphilitic hypothesis, he mentions that he has observed some cases complicated by genuine tertiary syphilitic lesions, which quickly disappeared under antisyphilitic treatment, whilst the paralysis progressed in the usual way. He emphasises the fact of the essential uniformity both of the clinical picture and of the pathological anatomy of general paralysis, and, recognising the difficulty in making such uniformity harmonise with the view that the disease has numerous different etiological factors, expresses the opinion that it is possible that it may yet be demonstrated that these various causes simply prepare the soil for a single intoxication, perhaps of a bacterial nature; and in the facts recorded by

<sup>1</sup> *Trattato di Psichiatria*, chap. xxx.

<sup>2</sup> *Trattato delle malattie mentali*, chap. xiv.

my colleagues and myself regarding the evidence of constant infection by a diphtheroid bacillus, he sees a possible solution of the problem.

Professor Tanzi, whilst provisionally accepting the syphilitic hypothesis as the one most strongly supported at the time of writing, is obviously dissatisfied with it. Few, if indeed any writers upon mental diseases have displayed so fine a gift of critical analysis in handling scientific evidence. His examination of the question inevitably leaves in the mind of the reader the conviction that, whether the syphilitic hypothesis is in accord with fact or not, the evidence upon which it rests is extremely incomplete and of very doubtful value. He clearly shows the necessity of invoking other etiological factors. He points to features of the disease that prove it to be dependent upon the action of some poison that gradually accumulates and then becomes destroyed or eliminated. He shows that this poison cannot be attributed directly to syphilitic toxines, and that therefore it must be assumed that it results from a consecutive auto-intoxication. He recognises the unsatisfactory vagueness of this theory, and refers to the more positive evidence of the occurrence of a bacterial toxæmia of gastro-intestinal origin, brought forward in 1901 by Dr Lewis Bruce and myself, as well as to some observations of Idelsohn upon the defective bactericidal power of the blood-serum of the general paralytic.

On several occasions, either independently or in conjoint papers, I have contended that the rôle of syphilis in the etiology of general paralysis and tabes dorsalis is only that of weakening the general and local defences, and that these diseases must be dependent upon an active bacterial toxæmia. The facts that seem to me to support this view are briefly the following.

Only a small percentage of syphilitics ever become general paralytics or tabetics. General paralytics have been known to become infected by syphilis, and it is extremely improbable that this could occur if general paralysis were essentially a late manifestation of syphilis. General paralysis may develop during the secondary stage of syphilis, and this is inconsistent with the view that the disease is either a tertiary or a quaternary manifestation of syphilis. Numerous cases of general paralysis in which previous syphilitic infection could be reasonably excluded have been known to many competent clinical observers. Anti-

syphilitic remedies, so promptly efficacious in tertiary syphilis are useless or even harmful in general paralysis and tabes. Statistics showing the high percentage incidence of previous syphilis are quite inconclusive. They leave entirely open the question of the occurrence of a secondary bacterial infection of a different nature, predisposed to by the syphilitic infection, just as tuberculosis is predisposed to by a previous attack of measles. Some of the arguments used to support the syphilitic hypothesis, as, for example, that drawn from the fact of the occasional occurrence of conjugal paralysis or of conjugal tabes, would better support the view that general paralysis and tabes result from a special venereal infection distinct from syphilis. The syphilitic hypothesis is devoid of the support of a single fragment of experimental evidence.

The theory that the toxæmia of general paralysis is a secondary auto-intoxication, directly dependent upon the previous action of syphilis, does not accord with the clinical and pathological facts. These point most conclusively to a struggle between the defensive forces of the individual, on the one hand, and an aggressive bacterial foe on the other, and indeed furnish evidence that places it beyond question that the general paralytic suffers from an active bacterial toxæmia. I have already, in the first lecture, referred to the evidence in support of this view brought forward in 1901 by Dr Lewis Bruce and myself. I have also described how Dr M'Rae, Dr Jeffrey and I were led, in 1903, to advance the hypothesis that in the production of this bacterial toxæmia, which is beyond doubt a mixed bacterial toxæmia, a diphtheroid organism probably plays a predominant part, and how Dr M'Rae and I, continuing to test this hypothesis, have gradually ascertained fresh facts which seem to us to warrant the conclusion that general paralysis and tabes dorsalis are essentially dependent upon infection by a special diphtheroid bacillus. The chief facts have already been stated, and it remains for me now merely to summarise the evidence in support of the conclusion that the *bacillus paralyticans* is the specific etiological factor in the production of general paralysis and tabes dorsalis, and to describe the morbid processes to which it gives rise.

I would have it clearly understood that the question is, in the meantime, left an open one whether this bacillus is merely

an attenuated Klebs-Löffler bacillus or an altogether distinct micro-organism. The decision of this question is not in the least vital to the argument that it is to follow. At the same time, I would say that, in my opinion, the evidence bearing upon the point is such as to render it probable that it will eventually be determined that the organism is a special one.

The evidence that this bacillus is the specific etiological factor in these diseases is, in brief, as follows :—

A bacillus of this nature is, according to the results of our investigations, present in large numbers in either the alimentary or respiratory tract, or in both, and in the genito-urinary tract in all cases of advancing general paralysis. This bacillus has a thread form which has been found invading the walls of the respiratory or alimentary tract in five cases of general paralysis. It can be shown that this organism in its bacillary form invades the pulmonary tissues in cases of general paralysis, and that it is commonly the only micro-organism present in large numbers in the catarrhal pneumonic foci that occur in most of such cases dying in congestive attacks. A growth of a diphtheroid bacillus has now been obtained in cultures made from the brain post-mortem in ten cases of general paralysis out of twenty-four in which cultures were made from this organ. Diphtheroid bacilli exhibiting metachromatic granules in Neisser preparations have been detected in the fresh blood in one case and in sections of the brain in two cases. It has been ascertained by experimental methods that these diphtheroid bacilli in contact with the living blood are rapidly taken up by the polymorpho-nuclear leucocytes, and that they may be completely digested in the course of two or three hours. Bodies exactly corresponding in appearance to these dissolving bacilli can be detected in the blood and cerebro-spinal fluid of the living general paralytic, especially during a congestive attack. Whilst the fact that most of the bacilli present are in process of disintegration satisfactorily explains the long succession of negative results of endeavours to obtain cultures from the blood and cerebro-spinal fluid, we have, by the use of special methods, succeeded in obtaining pure growths of a diphtheroid bacillus from the fresh blood in four cases of general paralysis, and from the cerebro-spinal fluid withdrawn by lumbar puncture in two cases. In sections of the brain prepared by special methods, disintegrating diphtheroid bacilli can be recog-

mixed in the walls of the vessels and in the pia-arachnoid in many cases of general paralysis. The centrifuge deposit from the urine of the general paralytic, especially during a congestive seizure, commonly contains abundant diphtheroid bacilli that have been more or less affected by lysogenic action (Fig. 6). In seven consecutive cases of general paralysis combined with tabes, we have found the centrifuge deposit from the urine to contain not only these altered diphtheroid bacilli, but also living ones showing distinct metachromatic granules in preparations stained by Neisser's method. In such cases a culture of the bacillus can be obtained from the urine. Experimental infection of three rats and a goat with diphtheroid bacilli isolated from a case of general paralysis has resulted in the production of symptoms and tissue-changes resembling those of general paralysis. Lastly, there is evidence that the active polymorpho-nuclear leucocytes of the general paralytic have, as a rule, a greater power of dissolving these diphtheroid bacilli than that possessed by the normal leucocyte. It would, therefore, appear that the general paralytic has acquired against these diphtheroid bacilli a certain degree of specific immunity, by means of which he is enabled to maintain the struggle against these bacilli, notwithstanding an otherwise defective local and general power of resistance.

I have next to answer the question, Does the view that this bacillus is the specific etiological factor in general paralysis and tabes dorsalis accord with the known clinical phenomena and the ascertained facts regarding the pathological anatomy of these diseases? In my judgment it does. I shall first sketch the pathogenesis of general paralysis as it appears to me in the light of the facts that have been ascertained, and afterwards I shall in a similar way consider the pathogenesis of tabes dorsalis.

The specific bacillus would appear to be conveyed from individual to individual by contagion, although there are grounds for believing that less direct methods of infection are not uncommon. It is capable of living as a saprophyte at the surface of the various mucous membranes. There is ample warrant for the conclusion that, to a person whose general and local defences against bacteria are intact, the *bacillus paralyticans* is quite innocuous. It can neither multiply to any important extent upon a healthy mucosa nor penetrate into the subjacent tissues. The case is, however, different if the bacillus becomes implanted



upon the surface of a mucous membrane that has been damaged in consequence of a long-standing catarrhal process, and more especially if the general defences of the individual have also become impaired. In normal conditions, epithelial surfaces, such as those of the respiratory and alimentary tracts, are protected from bacterial attack by a delicate layer of mucus, which has been shown to have not only a mechanical action, but also to be powerfully bactericidal (Arloing). As the result of certain forms of prolonged chronic catarrh, the function of the mucous glands tends to become exhausted; various saprophytic bacteria, normal or occasional inhabitants of the mucous tracts, are then liable to assume a pathogenic rôle. They do so, either in consequence of their excessive development at the surface of the mucosa and the absorption of their toxic products, or by actual invasion of the tissues. In cases of general paralysis a condition of severe chronic catarrh is constantly present, either in the alimentary or respiratory tracts, or in both. The mucous glands show marked signs of exhaustion, and there is excessive development of the common saprophytic bacteria. There are also good grounds for believing that the general paralytic, before he manifests signs of his disease, has suffered some impairment of his general defensive forces. There is, at least, almost constantly a history of his having been subjected to conditions that are known to cause such impairment. There are very numerous inimical forces that may produce such a condition of impaired local and general defence, but there are three that seem to have special importance in relation to general paralysis and tabes dorsalis. They are the pathogenic agent of syphilis, alcohol, and a too highly nitrogenous diet.

Syphilis is known to produce a severe drain upon the leucoblastic function of the bone marrow, whereby the general power of resistance must be injuriously affected. It is also known that it frequently determines the occurrence of chronic inflammatory lesions of various mucous membranes, more especially those of the mouth, throat, and respiratory tract. In this relation, it is perhaps not without significance that it has been ascertained that the *spirochæte pallida* lodges within various epithelial cells, including those of the bronchi.

The prolonged excessive use of alcohol, it is now well established, not only impairs the general power of resistance to

bacteria, but leads to severe morbid changes in the gastro-intestinal mucosa.

The prolonged excessive use of nitrogenous foods has also a disastrous action upon the general power of resistance and upon the local defences of the mucous membranes, as has lately been demonstrated by the experimental researches of Dr Chalmers Watson.

One or more of these three causes of impairment of the general and local defences against bacteria are almost constant in the individual history of the general paralytic.

It is upon such damaged mucous membranes that the *bacillus paralyticans* is capable of effecting a permanent lodgment. The organisms multiply in the catarrhal secretion, and also find their way into the ducts of partially exhausted mucous glands. Such saprophytic infection may continue for a long time without leading to any important toxic effects. It is probably only when the state of the local and general defensive forces is such as to permit of the bacillus invading the tissues that the paralytic toxæmia becomes of any great intensity. Our more recent observations have led Dr M'Rae and myself to attach special importance to the bronchi as a seat of chronic infection, although there are many cases in which bacillary invasion can be shown to have taken place from the gastro-intestinal tract. As in the case of other local infections, a veritable battle is waged between the attacking organisms, on the one hand, and the defensive forces of the individual on the other. It is virtually a life and death struggle between the bacilli and the polymorpho-nuclear leucocytes. It is a conflict in which the leucocytes, after a long succession of victories, are ultimately defeated, for their power of renewal is limited, whilst that of the bacilli is virtually unlimited. Moreover, there is warrant for the belief that in the course of a struggle of this nature, extending over many years, the virulence of the bacillus, especially in respect of its power to invade, becomes gradually increased. Under certain conditions the defensive forces are temporarily placed at a disadvantage and the bacilli become more aggressive. At least one of these conditions has been ascertained experimentally. It has been found that lowering of the temperature four or five degrees below the normal almost completely suspends the power of the leucocytes to take up the *bacillus paralyticans*. It is, therefore, reasonable to believe that lowering

of the body temperature of the general paralytic is an important cause of aggravation of the bacillary attack. Local invasion manifests itself clinically in a congestive attack, characterised generally by rise of temperature, always by leucocytosis and aggravation of the mental and bodily symptoms. After a few days, or a shorter time, the defensive forces, stimulated by the attack, may gain the upper hand and repel the invasion. The invaders are locally seized by the leucocytes and other phagocytic cells and rapidly destroyed. Others are dissolved, not by intracellular digestion, but by the action of the bacteriolytic ferments derived from leucocytes that have disintegrated. Large numbers of the invading bacilli reach the circulation either by way of the lymphatics, or more directly through the capillary walls. They may be seen in the blood-stream in the neighbourhood of the infective foci and also in films made from the patient's blood. In the blood, they are likewise quickly seized by leucocytes and digested; but here, as in the infective focus, it is not always the leucocyte that wins. There is evidence that very many of these cells, after partially digesting a number of bacilli, succumb to the action of the bacillary toxins and disintegrate, or at least disgorge their captives. The bacilli thus liberated, and others that have escaped the leucocytes altogether, pass out from the blood-stream by one or other of at least two ways. One is through the capillaries of the kidney into the urinary tract, and the other is through the damaged endothelial lining of the cerebral vessels into the adventitial spaces and other channels that constitute the lymph-system of the brain. That micro-organisms which have reached the blood-stream are commonly excreted by way of the kidneys is now a well recognised fact. That it occurs in this infection has been demonstrated. Why these bacilli should also be capable in certain cases of penetrating the endothelial lining of the cerebral capillaries is certainly not very easy to understand, but that they do so we can show. It probably depends upon certain special structural features of the cerebral vessels, as well as upon chemiotactic influences which direct the bacilli towards the nerve-tissues, with which their toxins most evidently combine. The disintegrating bacilli lodge for the most part in the adventitial lymph-channels, but many also reach the pia-arachnoid and subdural space. Within the lymph-channels of the brain, various endothelial and connective

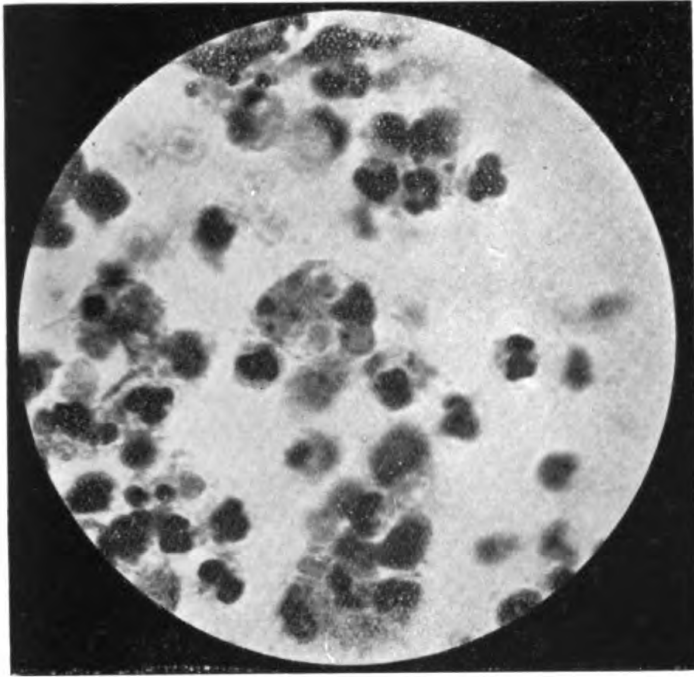


FIG. 8.

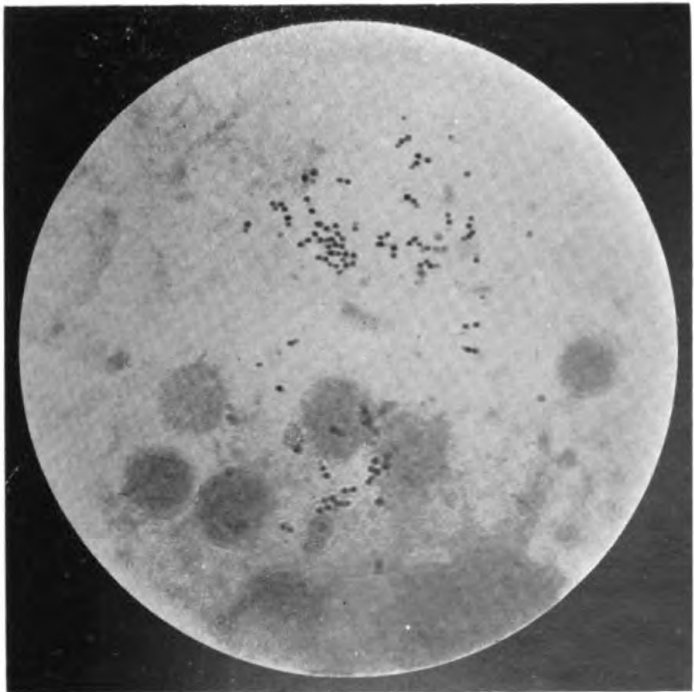


FIG. 9.



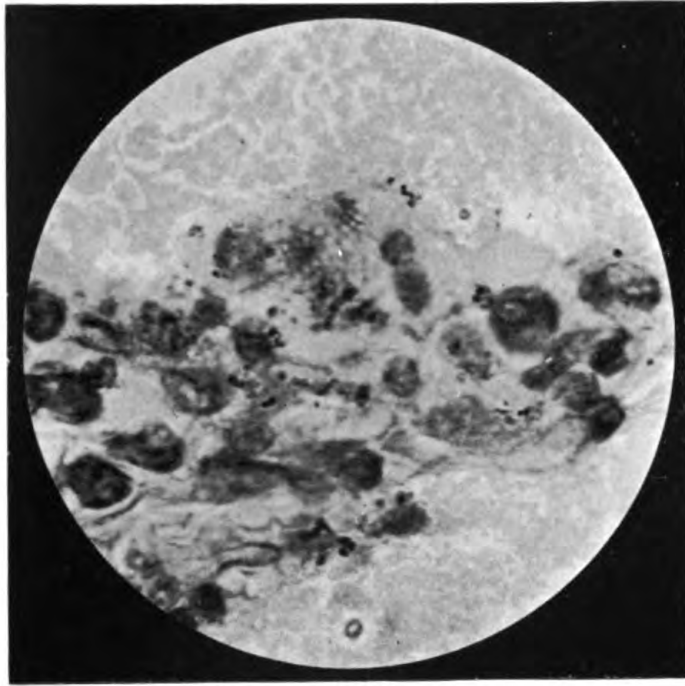


FIG. 10.

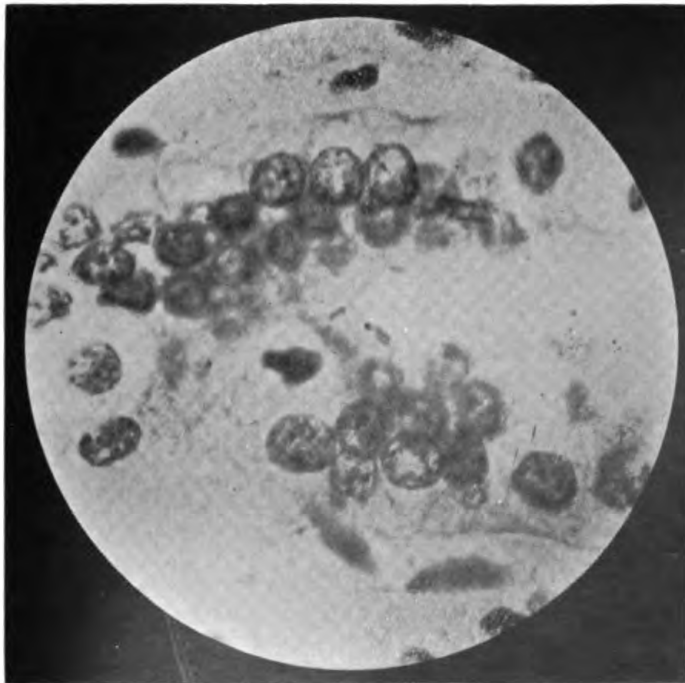


FIG. 11.



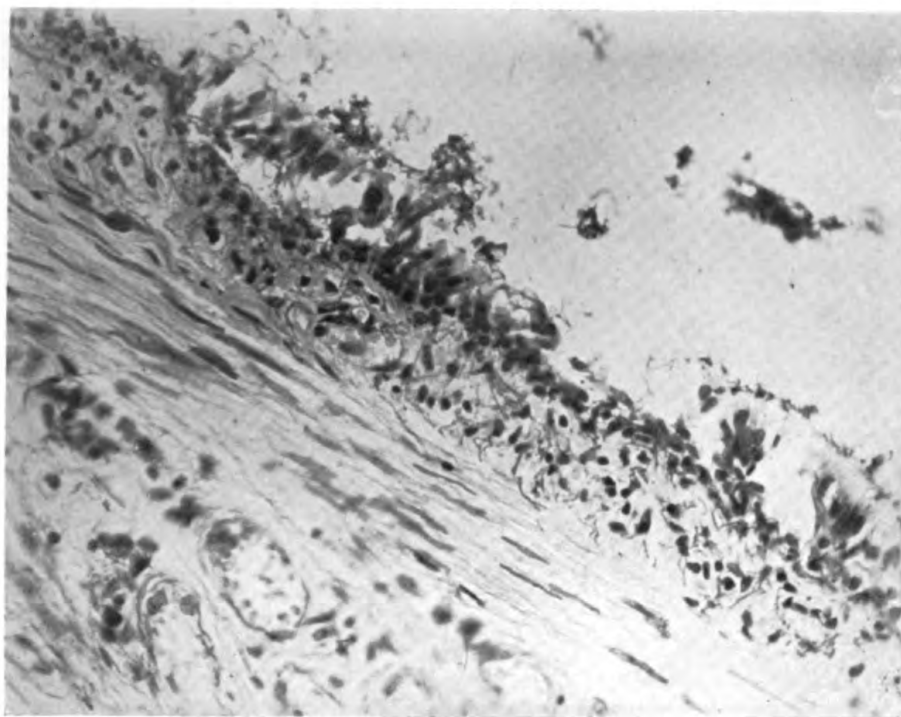


FIG. 12.

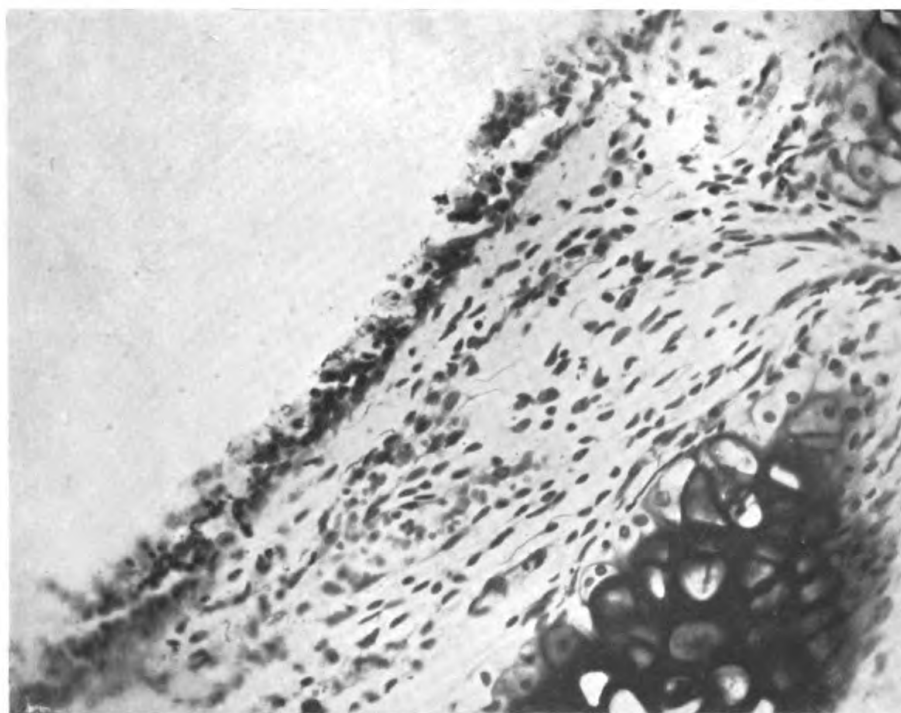


FIG. 13.





tissue elements exercise a phagocytic action and complete the destruction of the bacilli. These micro-organisms have been seen within lymphocytes in the centrifuge deposit from the cerebro-spinal fluid. The disintegration of the bacilli is attended by the formation of intense toxines. There is thus a general toxæmia resulting from the disintegration of the bacilli at the seat of invasion and in the blood, and an added local toxic action in the nervous centres, in consequence of the passage of the partially disintegrated bacilli through the endothelium of the cerebral vessels. The pathological changes that occur in the nervous system, already briefly described in the first lecture, are the result of this general toxæmia and local formation of toxines.

In some instances a successful repulsion of an invasion is followed by a prolonged period in which the bacillus is kept at bay. Clinically this corresponds to a remission. More commonly, however, there is a continuous comparatively slight absorption of toxines from the infective focus and a succession of more or less severe invasions, which time after time are repelled; in the end, however, the defensive forces are generally overcome. There is then a fatal congestive attack. General paralytics may, of course, die in other ways. In patients who die in congestive attacks there is commonly a more or less extensive catarrhal pneumonia. The catarrhal exudation is loaded with diphtheroid bacilli in various stages of disintegration. In other instances there is similar invasion from the stomach or small intestine. If pyrexia precedes death for some days, the bacillus tends to assume its thread form.

I come now to the subject of tabes dorsalis. That general paralysis and tabes dorsalis are in some way intimately related to each other is now so generally accepted that I need not argue the question here. On the anatomical side, apart from the systemic lesions of the posterior columns, there is in tabes evidence of a toxic action in the cord similar to that which occurs in the brain in general paralysis. There are two types of cases that especially require to be considered. There are cases of general paralysis which towards the end develop signs of tabes dorsalis, or at least show in the spinal cord the early changes characteristic of this disease, as indeed most cases of general paralysis do; and there are cases of pure tabes dorsalis

which, clinically at least, are not complicated by dementia paralytica.

I think we are greatly helped to a right understanding of this matter by the observations recently made by Drs Orr and Rows<sup>1</sup> upon the production of tabetic lesions of the cord. They have found that a systemic lesion affecting the posterior columns can be produced as a result of absorption of toxins from a peripheral septic focus, such as a bed-sore, and that there is distinct evidence that the toxins pass along the perineural sheaths of the nerves to the spinal cord. The peripheral nerve fibres remain unaffected, being protected by their neurilemma sheath, but the toxins injure the nerve fibres of the posterior root at the point at which they lose their neurilemma sheath, that is to say, as they pass into the cord. This is the vulnerable point of Obersteiner. They have also shown that the posterior column lesions in general paralysis, which are simply early tabetic lesions, start at the same point. These observations of Drs Orr and Rows, which are founded upon experimental evidence, as well as upon evidence drawn from cases, have confirmed the view long advocated by Obersteiner, Redlich, Dr Alexander Bruce and others, that in tabes dorsalis the systemic lesion begins where the posterior root enters the cord. In order to account for these lesions of the posterior root in general paralysis and tabes dorsalis, it is necessary to ascertain the source of the toxins that produce them. The morbid changes can in part, but not fully, be accounted for simply by toxicity of the spinal lymph. A peripheral toxic focus in close relation to the pelvic retro-peritoneal tissues is also required. In cases of general paralysis in which tabes supervenes, the toxic lesion of the posterior roots is, I think, in most cases sufficiently explained by the passage of disintegrating diphtheroid bacilli from an infective focus in the respiratory or alimentary tract through the blood and kidneys. In the urinary tract, further disintegration of these bacilli occurs, and the toxins thus produced are in part absorbed, and, entering the sheaths of the nerves, pass up the lymph-channels and so reach the vulnerable point of Obersteiner. In cases of pure tabes, in which there is an extreme degree of the same systemic lesion of the cord as that which occurs in almost every case of general paralysis, the

<sup>1</sup> *Brain*, Winter 1904; *Review of Neurology and Psychiatry*, January 1906.

source and nature of the toxins causing this lesion should be similar to those in general paralysis. In seven consecutive cases of tabes dorsalis we have found the centrifuge deposit from the urine to contain abundant unaltered diphtheroid bacilli, showing distinct metachromatic granules. Dr David Orr has at my request kindly sent me films of the centrifuge deposit from one case of simple tabes and from two cases of tabo-paralysis. In all of these three cases the films also show abundant diphtheroid bacilli with metachromatic granules. In these ten cases at least there was therefore what may be termed a diphtheroid cystitis. Whether this condition is constant in tabes dorsalis or not I cannot say, but if further observations should confirm the testimony of these ten cases, then I think we shall be bound to conclude that in tabes dorsalis there is in the urinary tract an infective focus comparable to that which occurs in the respiratory or alimentary tract in general paralysis. The bacilli are invading, and therefore produce toxic effects far in excess of those that result from simple passage of disintegrating bacilli through the urinary tract. We have had an opportunity of examining only one case of this kind post-mortem, and this so recently that there has not been time to make a complete histological examination; but, so far as we have been able to study the case, it bears out completely the view that in such cases a diphtheroid bacillus has obtained a hold upon the mucosa of the urinary tract. I may mention that in this case we readily obtained a growth of a diphtheroid bacillus from the urine; some weeks later the patient had a congestive seizure and we obtained a pure culture of the same bacillus from his blood; the congestive attack proved fatal, and we again obtained the bacillus in cultures from the brain. Sections of the bladder show the thread form invading in characteristic fashion.

There are two supplementary points that I wish to deal with very briefly. There are probably many who will find a difficulty in the way of accepting these views in the fact of the frequent presence of diphtheroid organisms in the alimentary, respiratory, and other mucous tracts of persons who are not suffering from general paralysis. I would recall such facts as that the Klebs-Löffler bacillus in its virulent form may not infrequently be found in the throats of healthy persons, and that the pneumococcus, which is the most common cause of pneumonia, can often

be isolated from the saliva of people who are perfectly well. I have already sufficiently insisted upon the importance of alterations in the local and general defences as a preliminary to the pathogenic action of the *bacillus paralyticans*, and what I have said should, I think, be a sufficient answer to this objection. It should also, however, be borne in mind that many of the diphtheroid bacilli that occur in other conditions may have much lower virulence than the micro-organism which we assert to be the essential etiological agent in general paralysis.

It has lately been shown that many persons who do not, strictly speaking, suffer from *tabes dorsalis*, nevertheless show many of the physical signs of this disease. Dr M'Rae has made observations which seem clearly to prove that a similar statement may be made in regard to general paralysis. It is only the more severe cases of this disease that are commonly recognised. Very numerous other persons are apparently infected by the specific bacillus, but resist it successfully, and show only very slight signs of having suffered from the paralytic toxæmia.

If the etiology and pathogenesis of general paralysis and *tabes dorsalis* are such as we assert them to be, how are these diseases to be combated? It is obvious that certain measures, such as are taken in other chronic infective diseases, should be adopted to prevent, as far as possible, the transmission of the bacillus to susceptible persons. I do not, however, wish to say more upon this point at present. Of more immediate interest is the question whether or not there is any reasonable prospect of these hitherto incurable diseases becoming amenable to treatment. On the ground of facts observed, I feel justified in saying, with considerable confidence, that there is. The general paralytic defends himself, and often with prolonged success, by manufacturing specific bacteriolytic anti-bodies, with the aid of which the invading bacilli are repelled. Such specific anti-bodies can be produced in suitable lower animals and used as therapeutic agents, and it seems probable that with their aid it may be possible to induce a prolonged remission of the paralytic toxæmia. If this could be effected at an early stage of the disease, the damage to the nervous system would be slight, and the result might legitimately be regarded as a cure. Dr M'Rae and I have for a considerable time been anxious to attempt to produce an anti-serum of this kind, but hitherto the difficulties

in the way have been insuperable. Thanks, however, to the energy of Dr Clouston, who has ever been ready to do all in his power to facilitate these researches, and whose unceasing interest in them has been one of the chief encouragements in our work during the last four years, these difficulties have at length been overcome. We are, at least, going to give such serum treatment a trial.

Allow me to make three acknowledgments before I close. I wish to express my indebtedness to Dr Harry Rainy for the great trouble he has taken in connection with these lectures, more especially in regard to the arrangements for the microscopical demonstrations. I have also to thank Dr M'Rae for so kindly relieving me of all trouble in connection with the laying out of the microscopical preparations; and, lastly, I desire to acknowledge my great indebtedness to the General Board of the Laboratory of the Scottish Asylums, that is to say, to the superintendents of the Edinburgh, Glasgow, Dumfries, Aberdeen, Perth and other Scottish Asylums, as well as to the lay members of the Board, for the facilities they have afforded my colleagues and myself for the carrying out of these investigations.

#### DESCRIPTION OF FIGURES.

- FIG. 8.—Cell in alveolus of pneumonic lung showing two diphtheroid bacilli in vacuole. From a case of general paralysis. Death in a congestive attack. Carbol thionin.  $\times 1000$ .
- FIG. 9.—Group of diphtheroid bacilli in blood-film taken from a general paralytic suffering from a slight congestive seizure. Neisser's method.  $\times 1000$ .
- FIG. 10.—Section of brain from a case of general paralysis, showing, in the adventitial lymph spaces of a cortical vessel, partially dissolved micro-organisms, which, in the microscopical preparation, can, in many instances, be recognised to have the characters of diphtheroid bacilli. Acidulated methylene blue.  $\times 1000$ .
- FIG. 11.—Section of kidney from a case of general paralysis. Shows, in a convoluted tubule, micro-organisms morphologically identical with diphtheroid bacilli, slightly altered by lysogenic action. Carbol thionin.  $\times 1000$ .
- FIG. 12.—Section of bronchus from a case of general paralysis. Shows the thread form of the diphtheroid bacillus invading. Carbol thionin.  $\times 600$ .
- FIG. 13.—Section of bronchus of rat fed for several weeks with bread mixed with unsterilised cultures of a diphtheroid bacillus isolated from the bronchus shown in Fig. 12. Shows similar invasion by the thread form of the diphtheroid bacillus. Carbol thionin.  $\times 600$ .

## **A CASE OF CYSTICERCUS CELLULOSAE OCCURRING IN THE INSANE.**

By H. EGERTON BROWN, M.D.,

Assistant Medical Officer and Pathologist, West Koppies, Pretoria.

THE following case of *cysticercus cellulosæ* may be of interest, partly on account of the large number and distribution of the embryos, and partly on account of the rapid progression towards amentia of the mental state of the patient, directly brought about by the same.

P., a Kaffir, was admitted to West Koppies, Pretoria, in October 1901. Physically, he was a fairly developed man and no evidence of disease was detected. Mentally, he was a simple dement with occasional outbursts of automatic violence; a typical example of this very common, or possibly most common form of mental disease among the native races of this country. In August 1902 he had two fits of an epileptiform character. During the last two months of his life it was noticed that a number of tense elastic swellings had appeared under the skin; some of these were excised and found to contain typical embryos of *tænia solium*. During this time he became rapidly more demented, and towards the end he bordered on a state of complete dementia, was unable to converse, took practically no interest in his surroundings, and was wet and dirty in his habits. The fits increased in numbers, acute phthisis supervened, and he died on October 13th, 1905.

Before describing the pathological appearances, it may be as well to point out the bearing the condition had on the patient's mental state. The epileptiform seizures were evidently not in the first instance caused by the embryo, although latterly, when the fits increased in number and severity, these were brought about without doubt by the cortical irritation of the growing cysts. As regards the mental state proper, I have the authority of Dr P. E. Todd, who has studied mental diseases among the natives for many years, for stating that this common form of insanity takes the form of a very slowly progressive dementia, and that it never, unless there be some concomitant pathological condition, reaches a condition bordering on amentia under eight

to ten years following the onset of the primary dementia. The depth of the dementia in this case is not to be wondered at when we consider the large amount of brain substance which has been destroyed and replaced by the cysts, and it is certain that one of the commonest causes of amentia, namely destruction of some of the higher association centres or their connections, must have taken place. Another point on which I base my opinion that the great exacerbation of the mental symptoms was directly due to the embryos, is the fact that these latter were all recent, as none of their cyst walls were in any way calcareous.

As regards the mode of infection, the theory of regurgitation of proglottides of the adult worm into the stomach, followed by the liberation of the ova by the acid secretion of the stomach, may be dismissed, as no signs of the adult worm were found either during life or in the intestines at the autopsy. The more probable cause was direct ingestion of some ova into the stomach by the mouth, as the adult worm is of common occurrence among the natives. (No further cases have occurred in over 200 natives living under exactly the same circumstances since the above date.)

*Autopsy.*—The body was much emaciated, and it was noticed that lying immediately under the skin were large numbers of small, tense, elastic swellings, 1 to 1·5 cm. in diameter; these, as have already been stated, were found to consist of cysticerci, which on examination showed the typical heads of *tænia solium*. On examination of the voluntary muscles, it was at once noticed that lying between, and in the same direction as the muscular fibres, were large numbers of the cysts, which in this position assumed a more elongated form than elsewhere. The pectorals and deltoids appeared to be the most affected.

On removal of the calvarium, nothing abnormal was detected, but on cutting through and raising the dura, it was noticed that the cerebro-spinal fluid was in excess, and that several typical cysts fell away, but four large ones remained firmly attached to this membrane, the largest measuring 2 cm. in diameter; this was evidently one of the so-called racemose type. On removal of the pia-arachnoid, as many as forty cysts fell away, while quite twice this number remained attached to the membrane; in places it was noticed that these left small depressions in the cortical substance; here as elsewhere the cysts were not arranged in any



definite relation to the blood-vessels. In no place was the pia-arachnoid adherent to the brain substance.

In the cerebrum, scattered all over the surface were numbers of the embryo, on the surface of the left hemisphere as many as seventy could be counted; here and there the cysts stood out from the surface in such a way as to cause the walls of the fissures to bulge apart; in others they were deeply embedded in the cortex, giving the appearance of small dark dots on the surface. It may here be stated that no one region of the cortex suffered more than another. On cutting transversely through one of the hemispheres, it was seen that the cysts occurred in great numbers, and although they tended to keep towards the surface, they were still to be found among the basal nuclei. In the photograph of the above the invaginated heads can be well seen. The greatest number to be seen in any one of these sections was twenty-four.

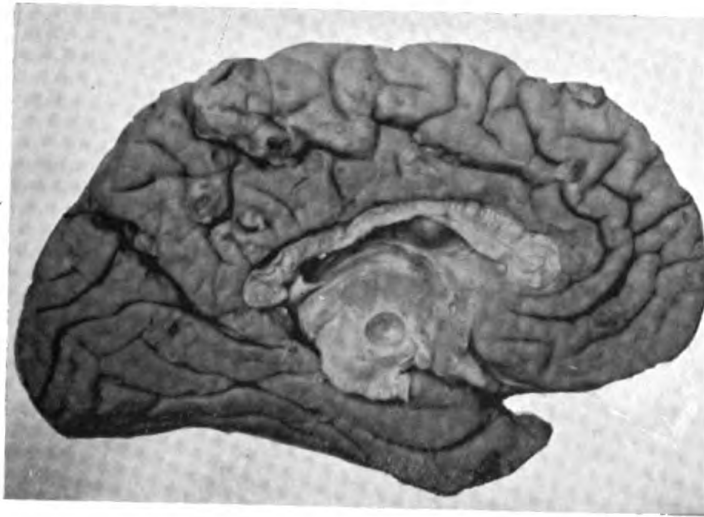
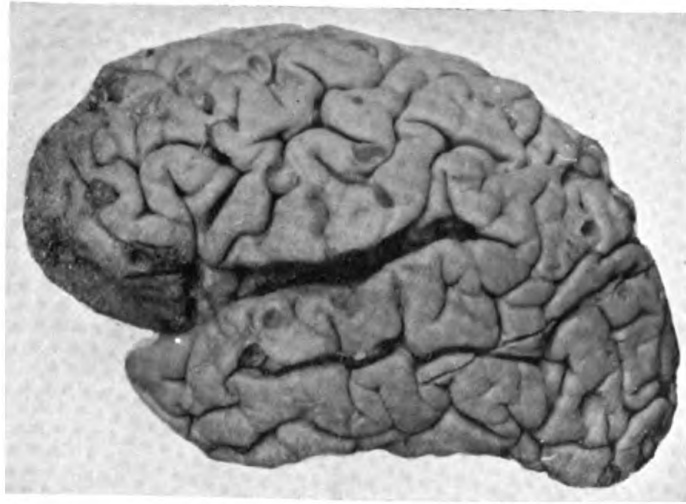
As regards the lateral ventricles, a few cysts were to be seen deep in the walls, but not projecting into the ventricles proper; on the left side, however, a very large cyst was to be seen projecting well into the ventricular cavity; this was the largest found in the whole body, and measured quite 3 cm. The choroid plexuses were free from embryos.

The cerebellum was in the same state as the cerebrum, but fewer embryos in proportion to its size were to be seen. The fourth ventricle contained no cysts. There were two large cysts in the pons, and these could be seen forcing the grey bundles apart. The medulla contained only one cyst, which was situated just above the decussation of the pyramids.

The spinal cord was free from cysts. On excising and making sections of the eye-balls, no cysts were found either before or behind the retina.

On opening the pericardium, no cysts were to be seen on the distal surface, but projecting from, and firmly adherent to, the apex of the heart, one of the racemose type was seen, showing, as has been pointed out in "Allbutt's System of Medicine," that this form is probably due to the condition of pressure under which it grows. On opening the heart, numerous cysts were to be seen peeping out from between the muscular bands; and in a place where one of the musculares papillares was cut through, the whole internal substance of the same was occupied by a cyst.

PLATE 20.





The diaphragm, lungs, pleura, kidneys, spleen, liver, and the intestinal walls were free from embryos. The intestines contained no tænidæ.

In conclusion, I have to thank Dr P. Everard Todd, Medical Superintendent, West Koppies, for permission to publish the above case.

---

## Abstracts

### PHYSIOLOGY.

**DEGENERATIVE SECTION OF THE NERVES TO THE CAT'S (124) BLADDER.** T. R. ELLIOTT, *Journ. of Physiol.*, Feb. 5, 1906, p. 29.

THE author, working in Langley's laboratory, found that division of the inhibitory nerves of the bladder, whether inferior splanchnics (preganglionic) or hypogastrics (postganglionic), produced in each case the same effect, viz. diminished size of the viscus and increased tone of its musculature, and this tone was not, as in the normal animal, at once depressed by section of the pelvic motor nerves.

After preganglionic section of the motor nerve (*nervus erigens*) on one side, the half of the bladder affected soon regained tone, and in the first week inhibition could be shown by stimulation of the hypogastric of the same side. This inhibition was not affected by the injection of nicotine, showing that the cell stations of these inhibitory fibres lie in the inferior mesenteric ganglia. A fortnight after section, stimulation of the motor nerve of the other side caused complete contraction of the whole bladder. This could not be elicited after the injection of nicotine, and the author thinks that it is to be ascribed to the outgrowth of preganglionic fibres from the sound trunk to the decentralised ganglia.

Preganglionic section of both motor nerves paralysed micturition, but there was always a good rhythm, which was at once checked by stimulation of the hypogastrics (inhibitory), and in a few weeks there was partial recovery. On the other hand, when the bladder was denervated by picking off the ganglia from its walls, the result was not the same as when the motor nerve cells remained; the muscle now lost its rhythm and became immobile, and did not respond well to faradisation.

Arguing by analogy, the author suggests that the ganglion cells of the bladder (and other viscera) may correspond to the motor

nerve endings of striped muscle, and that section of a motor nerve to striped muscle may correspond to preganglionic section in plain muscle. For example, the external sphincter ani muscle (voluntary) after section of its nerves reacts easily to stimuli, and exhibits rhythmic movements just as does the bladder after preganglionic section of its motor nerves, but not after paralysis of its motor end organs. Again, nicotine and curare both paralyse ganglion cells and motor nerve endings, and Langley and Anderson have proved that the preganglionic nerves of the autonomic system are functionally interchangeable with the motor nerves to striped muscle.

SUTHERLAND SIMPSON.

**ON THE REACTION OF CELLS AND OF NERVE-ENDINGS TO  
(125) CERTAIN POISONS, CHIEFLY AS REGARDS THE RE-  
ACTION OF STRIATED MUSCLE TO NICOTINE AND TO  
CURARI** J. N. LANGLEY, *Journ. of Phys.*, xxxiii., 4 and 5,  
Dec. 30, 1905, p. 374.

THE hitherto generally accepted view that adrenalin and some other substances act on the nerve-endings of unstriated muscle is at variance with many of the observed facts. Lewandowsky found that suprarenal extract had its usual stimulating action on the iris, eye, and nictitating membrane three weeks after extirpation of the superior cervical ganglion, and Langley confirmed his observations and added others of a like nature. Dixon showed that apocodeine paralysed post-ganglionic nerve-endings and abolished the action of adrenalin, but not of barium chloride. Langley finds that many muscles innervated by the sympathetic system react to adrenalin when the nerve-endings are completely degenerated. The action, therefore, seems to be neither on nerve-endings nor on plain muscle directly. Elliott has proposed an explanation in the presence of a new substance in unstriated muscle, which he calls the "myoneural junction," and which is the structure stimulated by adrenalin. Langley believes that the substance acted upon by many poisons is a part of the peripheral cell, and not necessarily the junction of the nerve with the cell it supplies. The paper is the outcome of an attempt to solve this question.

Fowls were used for the experiments, and the contractions of one or both gastrocnemii registered graphically. Injection of doses of nicotine into a jugular vein always caused contraction of striated red muscle fibres—the gastrocnemii fibres are chiefly red—even when their nerve supply has been divided. Where the nerve supply is left intact, the nerve is paralysed by large doses of nicotine, but the further injection of nicotine still causes contraction and the muscle responds to direct excitation. The make of a

galvanic current causes partial relaxation of the prolonged contraction due to the nicotine. The injection of curari annuls the effect of nicotine: the two drugs are antagonistic. This looks as though they acted on the same protoplasmic substance or substances.

Langley believes that nicotine combines with one substance, but with different degrees of completeness, according to the amount of nicotine present. Each successive act of combination produces a stimulation, but diminishes the excitability. The effect of the nerve impulse is thus abolished, and when no further combination can take place the further administration of nicotine also fails to stimulate.

After complete degeneration of the motor nerves, nicotine gives even greater effects on the denervated muscles, and the antagonism of curari is less than usual.

Langley concludes that nicotine and curari act upon the muscle substance, and not on the axon-endings. They do not prevent response of the muscle to direct stimulation, so probably act, not upon the contractile part, but upon some accessory material which he calls the "receptive substance" of muscle. This receptive substance presents considerable differences in different muscles and in different animals. The embryonic muscle protoplasm probably forms several receptive substances responsive to different chemical stimuli. In all cells two constituents at least must be distinguished—a substance concerned with carrying out the function of the cell, and receptive substances liable to change, and capable of setting the chief substance in action. Further, nicotine, curari, atropine, pilocarpine, strychnine, and most other alkaloids, as well as the effective material of internal secretions, produce their effects by combining with the receptive substance, and not by an action on axon-endings or on the chief substance. The receptive substance is not developed solely in consequence of nerve union, and is not confined to the myo-neural junction. PERCY T. HERRING.

#### ON THE METABOLISM AND ACTION OF NERVE CELLS.

(126) T. H. SCOTT, *Brain*, Autumn and Winter 1905.

THE author endeavours in this paper to cast some light on the function of the chromophile material of the nerve cell by an examination for the presence of a similar substance in other cells. As a result he finds that the only other cells in which a substance is found in the protoplasm, with properties similar to those of the Nissl bodies, are those which form the strong proteolytic ferments of secretion, *i.e.* the chief cells of the fundus glands and the pancreas.

The Nissl bodies have in common with these ferment-forming cells a large amount of highly organised iron-holding nucleo-proteid in their cytoplasm, the Nissl substance corresponding in its characteristics to prozymogen. The neurosomes in nerve cells are morphologically homologous with the zymogen granules of gland cells. There has been observed an interdependence between the amount of Nissl substance and the number of neurosomes, because activity in nerve cells leads to a diminution in chromophile substance and increase in fuchsinophilous granules (neurosomes).

The nuclei of nerve cells, cells of fundus glands of the stomach and of the pancreas, resemble one another. There is much resemblance too in their cell action, in that they are all concerned in controlling changes in proteids.

On the above similarities the author concludes that nerve cells also act by a kind of proteolytic ferment. DAVID ORR.

**UEBER DIE MATERIALLEN VERÄNDERUNGEN BEI DER**  
(127) **ASSOZIATIONSBILDUNG.** Prof. GOLDSCHIEDER, *Neurol.*  
*Centralbl.*, Feb. 15, 1906, p. 146.

By association is understood the fact that when several impulses reach different points of the cortex simultaneously or closely related in time, the future occurrence of the one impulse may recall the others independent of the corresponding external stimulus. It seems that by the association of two impulses the tracts and fibres situated between the affected centres come to offer less resistance to conduction than the paths which connect them with other centres, so that if one of the impulses is again produced it does not radiate indiscriminately in all directions, but discharges itself predominantly towards the other centre, which it stimulates in the same way as did the previous impulse from the periphery.

There has been hitherto no definite conception of the physical basis of this process. For a time it was explained by the amoeboid movements of the dendrites, but apart from the fact that this is unproven anatomically, no such gross mechanical theory is acceptable. Our conceptions are better stated in chemical terms, and then it matters not whether the theory of continuity or of contiguity of the nervous elements is accepted.

Vital activity, according to Hering, leads to dissimilation of the protoplasmic molecules, assimilation follows during rest. Hering's theory is most easily applicable by the adoption of Verworn's Biogen-hypothesis. The Biogen-molecule is an

assumed complex chemical combination, in a part of which dissociation occurs during activity and restitution follows immediately. But, as Weigert has shown, nature is never content with merely replacing the loss in such a condition, but attempts to form an excess. (It is on this assumption that Ehrlich's Sittenketten theory rests.)

On the simultaneous stimulation of two related neurones there is considerable dissimilation, as the activity of each reacts on the other owing to the spread of the impulses from one into the other; and if there be a succession of such stimulations, an hypertrophy of the Biogen-molecules will occur at the points of junction or contact of the two neurones, and will form a bridge across which the future discharge of such impulses can more easily take place.

When a series of cells, as generally happens, is simultaneously excited, their contact points, with the hypertrophied Biogen molecules, form a contact-point-line which represents the physical basis for the facilitated spread of stimuli coming from different cells. By this means an impulse which reaches the one cell can easily affect the whole series, and it thus calls up in consciousness a picture of the associated impressions which were originally received.

The discharge potentiality of the Biogen-molecules may not be equal throughout the whole line. Therefrom results defective memory and association; but by the further hypertrophy of the Biogen-molecules from repeated stimulation these become more perfect.

GORDON HOLMES.

**THE PARALYSIS OF INVOLUNTARY MUSCLE. Part III. On (128) the Action of Pilocarpine, Physostigmine, and Atropine upon the Paralysed Iris.** H. K. ANDERSON, *Journ. of Phys.*, xxxiii., 4 and 5, Dec. 30, 1905, p. 414.

RECENT investigations do not agree as to the manner in which pilocarpine, physostigmine, and atropine act on the iris. Anderson has made a series of observations on the action of each drug when used separately on the decentralised and denervated muscle of the iris. The animals employed were cats, the iris being decentralised by division of the oculo-motor nerve in the cranial cavity, and denervated by removal of the ciliary and accessory ciliary ganglia. After decentralisation, pilocarpine produces greater constriction of the paralysed pupil; physostigmine has less action, but both drugs constrict it for a longer time. After denervation, pilocarpine gives an increased and abnormally long contraction; physostigmine, however, does not stimulate the denervated sphincter. Pilocarpine



can act, therefore, on the sphincter muscle substance, but physostigmine on the nerve-ending only.

After imperfect regeneration of an oculo-motor nerve, physostigmine restores the light reflex when it is not detected under normal conditions; pilocarpine does not. Physostigmine does not increase the excitability or conductivity of the ciliary nerves or ganglia, or of the regenerating oculo-motor nerve. Its action shows that the impulses are blocked chiefly in the ciliary nerve-endings during regeneration of the nerve.

The denervated sphincter, after several weeks or months, begins to respond to physostigmine; and though there is no response to light, or to stimulation of the ciliary nerves, the action must be due to a regeneration, the exact nature of which is uncertain.

The nature of nerve-endings is shortly discussed. Pilocarpine acts on a portion of the sphincter muscle which is not the contractile substance itself. Anderson suggests the term "myoneure" for that part of Elliott's "myoneural junction" which persists in the muscle after degenerative section of its nerve supply.

PERCY T. HERRING.

**THE LAWS OF ERGOGRAPHY, A PHYSIOLOGICAL AND**  
(129) **MATHEMATICAL INVESTIGATION.** (*Les Lois de l'Ergographie, étude physiologique et mathématique.*) Mlle. J. IOTYKO (of Brussels), *Ann. d'Electrobiol. et de Radiol.*, No. 4, 1905.

VII. *Sugar*.—A short account of the ergography of sugar is given as a preliminary, which shows that there are considerable individual differences in its effect.

Dr Ioteyko herself experimented with three subjects; in none of these cases did *eau sucrée* have any influence on the ergogram. She then experimented again after imposing a fast of twenty hours. In two cases there was still no result—an interesting fact, as it shows that even in these circumstances the liver is still able to furnish abundant glucose; hence it appears that ergographic fatigue must be due chiefly to poisoning by the products of combustion, and not to want of combustible materials.

In the third subject the fast took effect, and the administration of glucose resulted in an ergogram which showed more than double the amount of work recorded in the ergogram of inanition.

Sugar is known to be an aliment for the muscles, and not to be an excitant for the centres. Therefore when the constants of the mathematical equation to the sugar curve are calculated, a diminution of  $a$  and  $c$ , which represent respectively the action of the toxins and the exhaustion of the carbohydrates, ought to be

shown. In effect this diminution does take place, and hence the analysis of the curve serves to confirm the interpretation which has been put upon the constants.

VIII. *Anæmia of the Arm.*—The effects produced by anæmia are physiologically just the opposite of those produced by sugar. As the circulation is stopped, supplies of glucose are not brought by the blood; moreover the toxic substances are not removed, and in the absence of oxygen retain all their harmful force. Hence ergographic fatigue in anæmia will be distinguished from that with the circulation by a more rapid exhaustion of the carbohydrates and swifter progress in the poisoning process.

Four experiments were tried, the anæmia being produced by a strong rubber band round the middle of the arm.

When the constants of the equations to the curves obtained are calculated, it is found that  $H$  (height of first contraction) diminishes, while  $a$  (action of toxins),  $b$  (action of nervous centres), and  $c$  (consumption of carbohydrates) all increase. The diminution of  $H$  betokens that the available quantity of carbohydrates is diminished; the increase of  $c$  that their consumption increases. The increase of  $a$  denotes the increased rapidity of the toxic action. The increase of  $b$ , denoting the increased activity of the nervous centres, is explained by the well established fact that increased resistance in the muscles acts as an excitant to the centres, causing them to send stronger stimuli to the periphery.

Hence the behaviour of the constants is again in accordance with expectation, and the experiment further strengthens the mathematical theory of the writer.

IX. *Cafféine.*—The mechanism of the action of cafféine on the muscles is not certainly ascertained, some writers maintaining that it acts as an aliment, others believing that it affects the muscles only through the mediation of the nervous centres. This fact led Dr Ioteyko to apply her mathematical method to the elucidation of the cafféine curve.

Her general conclusion from her investigation goes to confirm the opinion of Parisot, who regards cafféine not as an aliment, but as an excitant of the nervous system, allowing the reserves of the organism to be called into play. If, however, the dose be a strong one, or if a longer period be allowed to elapse between the administration of the cafféine and the writing of the ergogram, then the action is quite different. A phase of paralysis of the centre sets in, hence the consumption of carbohydrates becomes slower and more gradual, so that the contractions become lower, but at the same time more numerous.

X. *The Right Hand and the Left Hand.*—To simplify the investigation only right-handed persons were examined, the conditions, owing to education, etc., being much more complex in the

case of left-handed persons. The distinctive characteristic of the right hand ergogram, as compared with that of the left, is that the average height of the contractions is greater. Two curves in which this characteristic was well marked were chosen, and their constants calculated. The conclusion arrived at is that the predominance of the right side in right-handed people is essentially muscular, and arises from the fact that on that side a greater quantity of carbohydrates is available. This fact may easily be explained by the greater bulk of the muscles, by their more active circulation, and by their training which enables them to observe the most economical conditions of labour.

With all these researches very full tables of figures are given, enabling the reader to reconstruct the curves and also to test the mathematical work. The average difference between the height of contraction observed and the height as obtained by calculation is given in many cases, and never exceeds a millimetre and a half, while as a general rule it is much less. Reproductions of many of the curves are also given.

MARGARET DRUMMOND.

**THE LAWS OF ERGOGRAPHY, A PHYSIOLOGICAL AND**  
 (130) **MATHEMATICAL INVESTIGATION.** (*Les Lois de l'Ergographie, étude physiologique et mathématique.*) Mlle. J. IOTAYKO (of Brussels), *Ann. d'Electrobiol. et de Radiol.*, No. 5, 1905.

XI. *Accumulation of Fatigue, or Residuary Fatigue.*—This section opens with a pretty full résumé of the facts bearing on the question of the seat of fatigue. Professor Mosso, the inventor of the ergograph, maintains, in his book on the subject, that fatigue is nervous in nature. Dr Iotayko believes, on the other hand, that muscular fatigue is peripheral in origin. With the view of establishing her point, she examined the constants in 37 curves, written by 9 different people, to illustrate "residuary fatigue." This means that several curves were written, the interval between them not being long enough to allow of complete restoration.

A practically constant feature of the curves is a decrease in the quotient of fatigue (*i.e.* the average height of the contractions). If we accept the theory, first advocated by Hoch and Kraepelin, that the height is more particularly the function of the muscles, the number more particularly that of the centres, this in itself would indicate that fatigue first attacks the muscles.

The constants of all 37 curves being calculated, the writer finds that the most interesting results are: (1) the increase of  $\alpha$ , showing that residuary fatigue is muscular in origin, and is due to an

accumulation of toxic products in the muscle ; and (2) the increase of  $b$ , showing that the nervous centres, far from showing fatigue, are, by the very inertia of the muscle, roused to greater activity. Thus in fatigue the toxic action of the products of combustion plays a much more important part than does the failure of the carbohydrates.

It should perhaps be pointed out that the testimony of the figures is not quite so uniform as the text would seem to assume ; as a matter of fact,  $a$  increases 18 times and  $b$  17 times out of a possible 28. We may observe, however, that every time but once they vary in the same direction.

XII. This concluding section is devoted to pointing out a plan for new researches, and to urging on all workers the importance and far reaching power of the mathematical method inaugurated by this paper. Thus, to give a single example, the writer points out that in the different forms of paralysis, medullary, cerebral, or peripheral, an examination of the constants (especially if the healthy side can be compared with the side affected) will allow the seat of the malady to be determined.

MARGARET DRUMMOND.

(1) SOME ILLUSIONS REGARDING REST IN ERGOGRAPHIC  
(131) WORK ; (2) THE MECHANICAL VALUE OF THE MENTAL  
REPRESENTATION OF THE MOVEMENT ; (3) THE  
INFLUENCE OF ORIENTATION ON ACTIVITY ; (4) THE  
EFFECT OF A STANDING POSITION ON ERGOGRAPHIC  
WORK ; (5) THE EFFECT OF PRELIMINARY IMMOBILITY  
ON THE WORK ; (6) ECONOMY OF EFFORT ; (7) VARIABLE  
EFFECT OF THE SLACKENING OF RHYTHM ON  
WORK. CH. FÉRÉ, *Comptes rendus des séances de la Société de  
Biologie*, T. lix. et lx., 1905.

THESE papers by M. Ch. Féré give an account of various series of experiments made with Mosso's ergograph.

M. Féré finds that in his own case several influences, which we would be apt to think might safely be disregarded, affect the ergogram, and that to a considerable degree. Thus markedly different results are obtained when the orientation is altered. A position facing east or west is more favourable to work than one facing north or south. Again, marked affection of the tracings follows an immobility of the arm previous to executing the work. The arm was placed in position and kept so for varying times ; thereafter twenty ergograms were written, a minute's rest intervening between every two. Five minutes' immobility increased the work

of the first ergogram somewhat, while fifteen minutes decreased it by about six kilogrammetres. In this second case, however, the ninth ergogram shows a greater amount of work than the normal first. After an hour's immobility no ergogram shows even one kilogrammetre of work, *i.e.* they are all lower than the twentieth ergogram when there is no preliminary immobility. The total amount of work is greatest after an immobility of ten minutes. Another series of experiments was tried to find how the positions of standing and sitting affect the amount of work. Twenty ergograms were written in succession in each position. The figures obtained show that the standing position favours a greater amount of work at the outset, but gives rise to more rapid fatigue, so that the twentieth ergogram standing is not much more than a tenth of the value of the twentieth sitting. Another very remarkable series of experiments shows that the mental representation of writing an ergogram immediately preliminary to the actual performance of the work may cause so much fatigue that the normal ergogram cannot be written until the interval necessary for rest has elapsed.

By another set of experiments the author shows that residuary fatigue may exist in the muscle even when the ergogram shows no sign of it. We generally consider that the muscle has been completely restored when a second ergogram exactly like the first can be written. M. Féré found that with intervals of rest of fifteen minutes he could write eight ergograms equivalent to one another, but the ninth gave only 4.41 kilogrammetres of work instead of the normal 9.4, while the tenth fell to 0.78. This shows that fatigue may be masked for a considerable time.

According to Maggiora, ergographic work may be prolonged indefinitely if ten seconds are allowed to elapse between the contractions. M. Féré finds that with this rhythm, total exhaustion supervenes in times varying, according to the weight, from 51 minutes to 81 minutes 40 seconds.

Further experiments on rhythm show that with a slower rhythm (15 seconds as compared with 10 seconds) the number of contractions and the work done decrease, whereas the average height of the contractions increases. This fact shows that occupations requiring few movements, but necessitating continuous attention, may give rise to fatigue just as surely as those which appear more active.

If the rhythm is slackened still more, the same process goes on with increased rapidity, the average height of the contractions beginning to decrease also. When the rhythm is very slow the attention is periodically relaxed, so that the fatigue must be due to another factor, namely immobility. On the other hand, if experiments with slow rhythms are tried when the muscle is

already fatigued, then more work results than when it is not. The influence of the rhythm is thus very variable and complex, and more investigation is required to elucidate it.

A series of experiments with "economy of effort," that is, the ergogram being stopped as soon as the sensation of fatigue appears, shows that thus increased work may be obtained without a proportional loss of time. This is in accord with the fact demonstrated by Mosso that the first part of the ergogram, though representing more work than the second part, produces less fatigue in the muscle.

MARGARET DRUMMOND.

**A FURTHER CONTRIBUTION TO THE STUDY OF MENTAL (132) FATIGUE IN SCHOOL CHILDREN.** Dr JOSEPH BELLEI, of the Board of Health, Bologna, Italy, *The Lancet*, Feb. 3, 1906.

THE question whether the custom of holding afternoon as well as morning school is a healthy one for the children is one which it behoves educational authorities seriously to consider. Dr Bellei affirms in the most emphatic way that "the work done by the children during the afternoon lessons is, on account of the great mental fatigue that it involves, of no advantage to their instruction, but is full of danger to their health." This conclusion he arrived at in 1900 as a result of a series of experiments on mental fatigue during forenoon and afternoon school by means of the dictation method. At that time the interval for rest was from 12 noon to 12.45 P.M. In 1905 a similar series of experiments was tried, the interval for rest being then from 12 to 2 P.M. The results in the two cases are so nearly identical as to show that the longer period of rest has really no advantage over the shorter. Moreover, "the fact that the quality of the school work is almost the same at 2.45 P.M. and at 3.30 P.M. proves that after three-quarters of an hour of afternoon lessons the children are so tired that their work is full of mistakes, and becomes only a little worse after another hour of lessons."

MARGARET DRUMMOND.

## **PATHOLOGY.**

**PROOF OF THE EXISTENCE OF CHOLINE IN THE CEREBRO-(133) SPINAL FLUID.** (Preuve de l'existence de la choline dans le liquide céphalo-rachidien à l'aide du microscope polarisant.) J. DONATH, *Rev. Neurol.*, Feb. 16, 1906, p. 145.

THE contents of the paper are identical with those of a paper by the same author published recently in the *Journal of Physiology*, Vol. xxxiii. No. 3, p. 311.

In previous papers the author had tried to show that choline is present in the cerebro-spinal fluid of patients suffering from certain degenerative diseases of the nervous system, and in the cerebro-spinal fluids of epileptics. Choline was supposed to be present if an alcoholic extract of the cerebro-spinal fluid gave a precipitate with an alcoholic solution of platinum chloride. Characteristic crystals could be obtained by recrystallising the precipitate. It was found, however, that ammonium and potassium salts, which give similar compounds with platinum chloride, are always present in the alcoholic extract.

In the present paper a new and more certain method of testing the cerebro-spinal fluid for choline is detailed. It is based on the fact that the crystals of choline platinum chloride, if crystallised out of water, are doubly refracting, whereas the corresponding potassium and ammonium compounds do not possess this property. By means of the polarisation microscope one can therefore distinguish between the crystals of choline platino-chloride, which show double refraction and chromatic polarisation, and the crystals of the platino-chlorides of potassium and ammonium. Whereas the crystals of the latter substances are not visible with crossed nicols, the crystals of the choline compound appear bright or coloured in the dark field. If now, by rotating the table of the microscope, the angle of the optical axis of the crystals with the plane of vibration of the polarised light is altered, the crystals which were previously bright disappear from view after a rotation through  $45^\circ$  and reappear again after a further rotation through  $45^\circ$ .

By means of this method the author examined the cerebro-spinal fluid of 27 cases of progressive paralysis, chronic myelitis, idiopathic epilepsy, hystero-epilepsy, tubercular meningitis, tabes dorsalis, syphilitic cephalalgia, etc. In most cases he obtained positive results.

W. CRAMER.

**RESEARCHES ON THE BLOOD OF EPILEPTICS.** B. ONUF and (134) H. LOGRASSO, *Amer. Journ. of Med. Sciences*, Feb. 1906, p. 269.

SEVERAL cases were subjected to daily blood examinations for a long period in order to obtain definite data as regards the conditions in the intervals and in relationship to the attacks. Many cases had to be rejected owing to night attacks or other factors which might obscure the issues. In one case answering all the requirements no change occurred in the behaviour of the erythrocytes. The leucocyte count showed fluctuations in addition to those of health. A leucocytosis may occur before a seizure, but a grand-mal seizure may occur without a preceding leucocytosis

There is no exact parallelism between seizure and leucocytosis, and an existing leucocytosis may reach its height at different periods in different seizures. The leucocytosis is, in part at least, independent of the seizures, and marked rises of the leucocyte count do not necessarily mean seizures. When the intervals between seizures are long, the fluctuations of the leucocyte count are slight and are concentrated around the period of seizure.

ALEXANDER GOODALL.

**A CONTRIBUTION TO THE HISTOPATHOLOGY OF CERTAIN  
(135) FORMS OF PSYCHOSIS ALLIED TO DEMENTIA PRECOX.**

(Contribution a l'histopathologie de certaines formes de psychoses appartenant a la Démence Précoce [Kraepelin].)

DE BUCK and DEROUBAIX, *Le Neuraze*, Vol. vii., F. 2, p. 161.

IN the eight cases of dementia precox examined, the authors found that the principal lesion in the brain consisted of a pigmentary degeneration, together with a gradual atrophy of the neurones, which resulted in the complete disappearance of the nervous elements. The layers of the cortex most affected are those of the large pyramidal and polymorphic cells.

The neuroglia showed a more or less pronounced reaction, which, however, bore no definite relation to the various grades and forms of dementia precox. The vessels were only slightly affected.

These lesions, the authors suggest, depend on an auto-toxic action on a predisposed soil. The auto-toxines act very much like the exogenous toxines, in that they produce in the neurones a gradual degeneration which begins as a chromatolysis and goes on to a complete atrophy and disintegration of the nerve elements.

From the anatomical point of view, the authors suggest that dementia precox resembles closely the exogenous toxic psychoses, and especially that form which is produced by alcohol.

It is also somewhat similar to that which is found in epilepsy, and in acute delirium and confusional insanity. In the last two, of course, the lesion is much more acute.

Anatomically, therefore, dementia precox should be included amongst the toxic parenchymatous cerebropathies, of which it is one of the chronic forms; while the cases of acute delirium and of confusional insanity represent the acute forms.

If this view be correct, we may say that the brain behaves like the other organs of the body, and that toxines acting on it produce regressive parenchymatous changes, which may be acute or chronic, and which are not accompanied by diapedesis, by primary neuroglial overgrowth, or by vascular change.



If these last-mentioned changes appear, the case must be placed amongst the organic cerebral lesions, such as meningitis and progressive paralysis.

The authors do not, however, approve of the division of these dementias into the "démences organiques" and "démences vésaniques" of Klippel and Lhermitte. They maintain, rather, that the anatomical changes found prove the unity of certain nosological groups, which exhibit differences of degree rather than of kind, and they also throw some light on their ætiology.

Finally, they suggest the following anatomo-clinical classification of the acquired dementias:—

1. Dementia secondary to encephalitis and to new growths.
2. Senile dementia.
3. Epileptic dementia.
4. Paralytic dementia.
5. Dementia, the result of exogenous toxines.
6. Acute dementia, acute parenchymatous cerebropathy.
7. Dementia precox, chronic parenchymatous cerebropathy.

R. G. Rows.

**THE CONDITION OF MOTOR NUCLEI AFTER LESIONS OF  
(136) THE PERIPHERAL NERVES; AND THE PHYSIOLOGICAL  
SIGNIFICANCE OF THE EDINGER-WESTPHAL NUCLEUS.**

(Ueber das Verhalten der motorischen Kerngebiete nach Läsion der peripheren Nerven und über die physiologische Bedeutung der Edinger-Westphal'scher Kerne.) L. BACH, *Centralbl. f. Nervenh. u. Psychiat.*, Feb. 15, 1906, p. 140.

ALTHOUGH it is generally recognised that destruction of a motor nerve leads to the ultimate disappearance of the cells from which it sprang, it is not an absolute rule; occasionally cells persist indefinitely after their nerves are cut; on the other hand, if the nerve be torn out its cells of origin almost invariably disappear.

After destruction or removal of an eye the third nerve nucleus remains quite normal, and the author contends that it must be so, as the ciliary ganglion, from which intrinsic muscles are supplied, is a sympathetic ganglion. As this has, however, been denied, the author undertook further experiments to prove his assertion. About seven months after the removal of an eye with a considerable portion of the third nerve, the midbrain was examined by Weigert's method, and it was found that the cells which supply the external ocular muscles were degenerated. In the oral part of the oculomotor nucleus almost all the cells in the same side had disappeared, while in the caudal end the loss was chiefly in the dorsal portion of the opposite nucleus. The Edinger-Westphal

nucleus was normal. After a critical survey of all the facts which can be obtained in the literature on the subject, the author comes to the conclusion that there is nothing to support the view that this nucleus is the centre for the sphincter pupillæ. Koelliker was probably right in not including this collection of cell in the oculomotor nuclei.

GORDON HOLMES.

## CLINICAL NEUROLOGY.

### FACIAL PARALYSIS AND HEMIATROPHY OF THE TONGUE.

(137) (*Paralysie faciale et hémiatrophie linguale droites ayant vraisemblablement comme origine un polioencéphalite inférieur aigue ancienne.*) E. HUET et P. LEJONNE, *Rev. Neurolog.*, fév. 16, 1906, p. 105.

THE patient was a girl of 15, who, at the age of 3 years, in the course of a febrile attack accompanied by convulsions, developed facial paralysis on the right side. Later, transient ocular weakness was observed, which passed off under electrical treatment. The facial palsy persisted unchanged till she came under observation. It was found to be flaccid in type, but with fibrillary contractions and occasional paroxysmal twitching of the right labial commissure. The palsy involved all the facial muscles, most markedly, however, those supplied by the middle branches of the facial, attacking the upper fibres less severely, and the lowest fibres least of all. The tongue was slightly wasted on the right side, but without motor weakness. The electrical reactions of the face and tongue were normal. No other cranial nerves were affected, and the limbs showed no abnormality of motor, sensory, or reflex functions.

The authors consider the phenomenon to be the result of an old acute nuclear affection—a polio-encephalitis inferior, implicating the facial and hypoglossal nuclei on the right side. The sudden febrile onset, with convulsion, supports such a hypothesis. They consider the fascicular contractions in the paralysed muscles as due to a degree of irritability in the cells of the facial nuclei.

PURVES STEWART.

**DU TABES TARDIF.** E. LONG and A. CRAMER, *Rev. Neurolog.*, Feb. 15, (138) 1906, p. 110.

IN tabetics of advanced age, we have to recognise two classes of cases: firstly, "prolonged tabes" in patients who, having presented the first symptoms of the disease at the ordinary middle period of life, have survived to old age; secondly, those in whom the disease

first attacks the patient in advanced years. This latter class of "late tabes" is the one to which the writers direct their attention.

Out of a series of 46 tabetics, 15 cases, or almost one-third, commenced after fifty years, and of these, 5 of them after sixty years. This proportion is not claimed to be the ordinary one, in fact it probably exceeds the ordinary figures.

Out of ten cases of tabes where a previous syphilis was certain or probable, in four cases the syphilitic infection preceded the first tabetic symptoms by from five to twenty years. In the other six, the incubation period of tabes was much longer, varying from 30 to 42 years.

In other cases, again, the lateness of the tabes was explicable by the lateness of the age at which the syphilis was acquired.

PURVES STEWART.

**A STUDY OF THE LARYNX IN TABES.** D. CROSBY GREEN, (139) jun., *Boston Med. and Surg. Journ.*, Jan. 25, 1906, p. 97.

THE author reports the result of recent examinations made by him in sixty cases of tabes. These were observed with reference (1) to the proportionate number affected with paralytic and other disturbances of the larynx; (2) to the nature of such disturbances; and (3) to the period of their occurrence in the course of the disease.

1. Out of the cases examined, 15 per cent. presented laryngeal complications; 10 per cent. showed undoubted paralysis of one or both vocal cords; 12 per cent. were affected with laryngeal crises, three of these without evident paralysis of either cord, and in one the cord moved from the median line in a jerky manner. (This latter figure is small as compared with that of Dorendorf, of Berlin, who found 4 per cent. with ataxic movements.)

2. Abductor paralysis was the only form seen. Of 6 cases, 5 were unilateral and 1 bilateral, being complete in 3 and only partial in 3. In this latter connection the author points out that partial abductor paralysis should not be diagnosed without repeated examinations, since physiological variations in the degree of the excursion of the vocal cords are met with. Contrary to Semon, Dorendorf, and others, he found no case where, owing to the rapid degeneration of the nerve, a complete recurrent paralysis followed the abductor paralysis.

In 4 cases, laryngeal crises were associated with paralysis, while in 3 there was no paralysis. In 1 case of bilateral abductor paralysis the crises, when of three years' standing, greatly diminished in frequency and severity, owing, the author supposes, to the weakening of the adductors.

Differing from others, Dr Green found no case of real disturbance of laryngeal sensibility.

3. The laryngeal crisis when present occurred among the earliest symptoms in all his cases, and in two of them (briefly described), owing to a complaint of spasmodic cough, it led to the examination of the larynx and the subsequent detection of the tabes.

He infers that laryngeal paralysis is an early symptom of tabes, but cannot say at what stage it occurs in the majority of cases.

The practical conclusion to be drawn from this study is that tabes as an etiological factor, either of vocal cord paralysis or of spasmodic laryngeal cough, should never be overlooked.

J. D. LITHGOW.

#### **LANDRY'S PARALYSIS AFTER ENTERIC FEVER: RECOVERY.**

(140) (*Acute aufsteigende [Landry'sche] Paralyse nach Typhus abdominalis mit Ausgang in Heilung.*) SCHUTZE, *Berlin. klin. Woch.*, Feb. 12, 1906, p. 201.

CASES of Landry's paralysis following enteric fever have been reported by Leudet, Curschmann, Kümmell, Pitres, and Vaillard, but the complication is a rare one, and is almost invariably fatal. It is diagnosed from meningo-myelitis and syphilis of the spinal cord by the absence of sensory symptoms and of muscular atrophy, and by the retention of control over bladder and rectum; and from ordinary polyneuritis, by the absence of pains, of electrical changes, and of muscular atrophy.

Schutze's patient was a soldier, aged 25, who was admitted to hospital with a severe attack of enteric fever in which hypostatic pneumonia and intestinal hæmorrhage subsequently developed. A week after the temperature had become normal he began to complain of a sense of weariness in the legs. The temperature rose again to 101°. The urine, hitherto clear, contained a trace of albumen, and the spleen became again enlarged and tender. A relapse was at first suspected, but there were no rose spots nor typical stools. The next day the patient complained of severe pain along the vertebral column, which was aggravated by touching or tapping the spinous processes, especially in the lumbar region. He was quite unable to move his legs or to sit up in bed. Passive movements revealed a flaccid paralysis of the muscles of the pelvis, abdomen, and lower limbs. Faradic excitability was preserved, but was somewhat diminished in the calf muscles of the left leg. The knee-jerks were lost on both sides. The plantar, cremasteric, and abdominal reflexes could be obtained, but were obviously

impaired. Except for a small zone of complete anaesthesia on the back of the right foot, tactile and painful sensation was quite unaffected. Slight convergent strabismus was present. The pupils were equal and reacted to light. The conjunctival and corneal reflexes were present. The sphincters were intact.

During the next two days complete flaccid palsy, first of the right and then of the left arm developed. The electrical reactions remained practically normal, and, with the exception already mentioned, there was no impairment of sensation.

On the fifth day the respiration became laboured and there was occasional Cheyne-Stokes breathing, but these signs of threatening respiratory paralysis disappeared the next day, when an ephemeral difficulty in articulation developed. There was no affection of the palate, lips, pharynx, or eyes, but a slight left facial paralysis of three days' duration occurred. After complete paralysis of the limbs, lasting for six days, power began to return, first in the arms and then in the legs. Within a week from the onset of the paralysis, the patient was discharged cured. There was no evidence or history of syphilis, but he was nevertheless subjected to mercurial inunction and the administration of potassium iodide internally for a fortnight before leaving hospital.

J. D. ROLLESTON.

#### **DISEASES OF THE CONUS TERMINALIS AND OF THE CAUDA**

(141) **EQUINA.** (*Über Erkrankungen des Conus terminalis und des Cauda Equina.*) R. BÁLINT and H. BENEDICT (Budapest), *Deut. Zeitschr. f. Nervenheilk.*, Bd. 30, H. 1-2, p. 1.

SIX cases are described in this paper: (1) traumatic injury destroying the cord from L5 downwards (confirmed by *sectio*); (2) primary neuritis (?) of 3rd, 4th, and 5th sacral roots; (3) spina bifida, with involvement of parts of the roots of L5, S1, S3, S4, and S5; (4) aneurism eroding through the sacrum and pressing on 4th and 5th lumbar and 3rd, 4th, and 5th sacral roots; (5) meningo-myelitis of S1, S3, S4, and S5; (6) neurofibroma of lower lumbar sacral cord.

Localisation of the lesion in the conus or cauda is not always easy, except in such cases as Nos. 3 and 4, where the site puts out of the question an affection of the cord. Irregularities in distribution of the sensory and motor disturbances are commoner in cauda cases than in lesions affecting the conus.

It is noteworthy that in case 4 there were fibrillary twitchings of the glutei, which, according to several writers, is purely a sign of a conus lesion, while this was undoubtedly a radicular case.

A full analysis of the urinary, defæcatory, and genital symptoms is given.

In every instance micturition was of the involuntary automatic type of childhood, but with more or less residual urine. In no case was there any real paralytic incontinence. The writers favour L. R. Müller's view that the automatic bladder centres are not in the cord, but in the pelvic sympathetic, but little light is thrown on the share taken by the lumbar and sacral parts of the cord in the innervation of the bladder.

Defæcation was quite involuntary, but despite the paralysis of the sphincter ani there was usually continence. That there is an automatic centre functioning independently of the spinal cord and lying in the sympathetic pelvic ganglia, is almost certain.

Little was learnt regarding the seat of the genital centres in the male, but the two female cases (1 and 2) are of interest. Case 1 had a labour entirely painless; it would therefore seem as if for the innervation of the body of the uterus (which, according to Head, is supplied by the lumbar sympathetic) the 5th lumbar rami were chiefly concerned. The centre for labour pains cannot be in the sacral cord. As with micturition a spinal centre (in the lumbar cord) cannot be entirely excluded, but taken in conjunction with animal experiments it seems probable that such a centre is confined to the pelvic sympathetic ganglia. Both patients, although ovulating normally, became absolutely frigid, apparently from the complete anæsthesia of the lower part of the genital passages.

J. H. HARVEY PIRIE.

**ANALYTICAL EXAMINATION OF THE SYMPTOMS AND ASSO-**  
**(142) CIATIONS OF A CASE OF HYSTERIA.** (*Analytische Unter-*  
*suchungen der Symptome und Assoziationen eines Falles von*  
*Hysterie.*) F. RIKLIN (of Rheinau), *Psych.-Neur. Wchnschr.*,  
 Nr. 46-52, 1905.

THIS is an important contribution to our knowledge of the mechanism of hysterical disorders, and is the result of two years' observation and analysis of the case recorded.

Patient was a hysterical girl, aged 23, of moderate intelligence and poor physique, who had had a most stormy life, full of revolting sexual episodes; she suffered the first sexual trauma at the age of 12, while menstruation began at 13. On the death of her third illegitimate child, she was sent, at the age of 23, to the house of correction for a year, was then admitted to a refuge for women, and, owing to depression and suicidal ideas, was admitted to the hospital for the insane.

Physically, nothing objective of any importance was made out, while patient had a great variety of subjective complaints, which were the important element in the course of the disease. Among these latter were severe pain in the side, cough, vomiting, pelvic and abdominal pains, inability to walk, pain in the ear. Examination of the patient in the hypnotic state showed that these symptoms were the representatives in consciousness of subconscious or submerged complexes of ideas connected with the various painful episodes of her life. Her persistent vomiting was due to the frequent associations which led to the submerged complexes, based on outrages which had at the time caused vomiting or nausea; patient herself was unconscious of this reason for the vomiting, as the associations were split off and did not form part of her conscious system of associations. For a long time milk produced nausea and was refused, and she herself gave plausible but superficial explanations: the real ground, as disclosed in the hypnotic condition, was the fact that during an early outrage in a barn a pail of milk was spilt; the incident, which in hospital caused patient to refuse milk, had led by association to this submerged complex without bringing it to light. In a similar way Riklin traced the mechanism of the other symptoms, and he shows how experiences accompanied by deep emotion, and connected, perhaps casually, with somatic symptoms, tend to be thrust into the background of consciousness or submerged, and thus remain as a foreign body or an irritant in the mind, having as their representatives in consciousness various disconcerting symptoms. When the hidden sore is touched, although the patient is unconscious of the mechanism, the conscious life is disturbed by an unexplained disorder. Under hypnosis the deeper explanation of the disorder is reached, and the ventilation of the split-off and submerged complex tends to remove its noxious effect. The confession of the whole episode enables the patient to digest and assimilate it, and this justifies the probing of the secrets of the patient against her will: thus, after the elucidation of the basis of her disgust for milk, patient lost her repugnance for it. This gastric complex was one of many physical complexes corresponding to a well-defined system of split-off complexes.

The analysis of patient's associations, tested by giving one word and asking what other word that suggested, showed that wherever the given word was related to a split-off complex the reaction was abnormal in time or in other ways. Such abnormal reactions enable one to come upon the trail of a hidden complex.

In conclusion, the author discusses the associative mechanism of various forms of hysterical disorders.

C. MACFIE CAMPBELL.

**THE CLINICAL HISTORY AND POST-MORTEM EXAMINATION  
(143) OF FIVE CASES OF MYASTHENIA GRAVIS. E. FAR-  
QUHAR BUZZARD, *Brain*, 1905, p. 438.**

DR FARQUHAR BUZZARD has examined post-mortem five cases of this disease, probably a larger number than has been examined by any single observer up to the present time. In this paper the clinical history of these cases and the histological findings are described *in extenso*.

It is impossible in an abstract to do justice to this valuable and interesting communication, which is one of the most important contributions on the subject which has hitherto appeared, and must of necessity be carefully studied by all those specially working at the pathology of the disease. The author summarises his conclusions thus:—

*Clinical*.—(1) That myasthenia gravis is a disease in which the symptoms are not always confined to the motor system, but may include others of sensory, mental, or other origin.

*Anatomical*.—(2) That in all probability it has a definite and constant morbid anatomy, constituted by the presence of widely distributed cellular, and sometimes serous, exudations (lymphorrhages) in the tissues and organs of the body.

(3) That slight muscle-fibre changes are frequent, and severe muscular atrophy rare, occurrences in the disease.

(4) That proliferative and degenerative changes in the thymus gland are frequently, but not constantly, met with.

*Theoretical*.—(5) That the symptoms of the disease are best explained by assuming the presence of some toxic, possibly auto-toxic, agent, which has a special influence on the protoplasmic constituent of voluntary muscle, and a less specialised influence on the function of other tissues.

(6) That the relation of this toxin to the incidence of lymphorrhages and to thymic alterations is not clear.

The two very beautiful plates illustrating the morbid appearances in the muscles are deserving of mention.

EDWIN BRAMWELL.

**THE ASTHENIAS AND MYOPATHIC ATROPHIES. (Asthenies  
(144) et atrophies myopathiques.) M. KLIPPEL and M. VILLARET,  
*Archiv. Gen. de Méd.*, Feb. 13, 1906.**

THE authors make an attempt to show that the whole group of atrophic and hypertrophic myopathies, myasthenia gravis, Thomsen's disease, and family periodic paralysis are not only intimately related but perhaps different manifestations of the same patho-



logical process. A study of their paper suggests that they commenced with this assumption, and then laboriously undertook the task of giving it some semblance of reality by hunting the literature for the most atypical examples of each disease, and pointing out certain points of resemblance to other members of the group. Such an undertaking might be considered bold if we were in possession of complete knowledge concerning the origin of each of these various morbid processes. In our ignorance of the exact nature of any one of them, we must regard this piece of work as distinctly premature and unsatisfying. Had Klippel and Villaret been content to intimate that, in their opinion, the diseases mentioned above were all dependent upon some form or other of disordered muscular metabolism, many observers would probably not be prepared to quarrel with them.

E. FARQUHAR BUZZARD.

**A CASE OF CRURAL MONOPLÉGIA, PROBABLY REPRESENT-**  
(145) **ING THE EARLY STAGE OF A UNILATERAL ASCENDING PARALYSIS DUE TO DEGENERATION OF THE PYRAMIDAL TRACTS.** C. K. MILLS, *Journ. Nerv. and Ment. Dis.*, Feb. 1906, p. 115.

THE case is that of a woman, 50 years old, with a history of gradually increasing weakness of the right leg. When seen 14 months after the onset, she had moderate paresis of the entire limb, more marked distally, greatly exaggerated knee-jerk, persistent ankle clonus, and Babinski response on the right side. There were no sensory symptoms and no other detectable nervous affection except (?) a very slight weakness of the right arm, and increased knee-jerk and muscle-jerks on the left leg. Mills believes that the case is best explained by degeneration of that portion of the pyramidal tract which passes from the motor cortex to the lumbo-sacral cord, and that it is altogether probable that the disease will advance and involve the arm of the same side and also the unaffected lower extremity. Reference is made to several similar observations of unilateral ascending paralysis, due to progressive degeneration of the pyramidal tracts—confirmed by autopsy in one case.

A. W. MACKINTOSH.

**SEPARATE SENSORY CENTRES IN THE PARIETAL LOBE**  
(146) **FOR THE LIMBS.** W. G. SPILLER, *Journ. Nerv. and Ment. Dis.*, Feb. 1906, p. 117.

SPILLER records the case of a man, 38 years of age, who, when examined 17 months after being struck over the right parietal lobe

with a club, exhibited the following condition: awkwardness and ataxia but no paresis of the left arm; great impairment of the sense of position, of the spacing sense and of stereognostic perception in the left hand, with slight diminution of the sensations of touch, pain and temperature in this hand; no affection, motor or sensory (subjective or objective), in left leg or face or on right side of body; no hemianopia; patellar reflexes not prompt. Paræsthesia had been present since the injury in the left hand, but had never occurred in left leg or face.

Spiller is inclined to place the lesion in the lower part of the parietal lobe and he believes that the case shows: (1) that sensation may be affected from a cerebral cortical lesion, without motor paralysis; (2) that the sensory alteration may be confined to one limb, *i.e.* that the sensory centre for the upper limb must be distinct from the centres for the face and lower limb; and (3) that the alteration of the sensations of position and space and of stereognostic perception is greater from a lesion of the parietal lobe than is the alteration of the sensations of touch, pain and temperature.

A. W. MACKINTOSH.

#### **HYSTERICAL STIGMATA CAUSED BY ORGANIC BRAIN**

(147) **LESIONS.** HERM. H. HOPPE, *Journ. Nerv. and Ment. Dis.*, Feb. 1906, p. 101.

NOTES of two cases of organic cerebral disease are given in which hysterical signs appeared. Remarks are made on the "psychology of hysteria." Hoppe agrees with Oppenheim that all the phenomena of hysteria can be explained by "an irritable weakness, an abnormal exhaustibility of function of the brain cortex"; therefore, "we may assume that the same protoplasmic changes of the ganglionic cells which we assume to be present in neurasthenia are also present in hysteria." "I assume that hysterical signs and symptoms which we see accompanying organic diseases of the brain are the result of organic changes in the ganglionic cells"—these changes resembling very closely the conditions found in exhaustion from functional activity, and being produced by such results of the organic disease as increased intracranial pressure and circulatory disturbances. The paper deals mainly with purely "theoretical considerations" and assumptions.

A. W. MACKINTOSH.

**ATAXIA IN CHILDHOOD.** FREDERICK E. BATTEN, *Brain*, 1905, (148) p. 484.

THE paper deals with ataxia in childhood. The author excludes from consideration ataxia occurring in association with Friedreich's

disease, with tumours or lesion of the cerebellum or mid-brain, and with certain cases of diphtheritic paralysis. He divides his cases into three groups—

(1) *Congenital cerebellar ataxia*.—Cases in which ataxia has been noted early in life and in which there is a tendency to gradual improvement.

(2) *Acute ataxia*.—Encephalitis cerebelli—cases in which ataxia has suddenly developed after some acute illness in a child who formerly had been quite healthy.

(3) *Progressive cerebellar ataxia*.—Cases in which a child has been healthy till a certain age, and then has gradually developed ataxia.

Two cases illustrative of the first group are given. Both children exhibited marked ataxia of gait, which, during the three years they were under observation, became less and less marked. There were constant movements of the head and trunk and marked inco-ordination of the hands. The articulation was jerky—a very characteristic feature of the disease.

Under the second group five cases are described—two of which were observed by the author, the remaining three by Dr Voelcker, Dr L. Guthrie, and Dr Frederick Taylor. In the one of these which may be taken as typical of the disease, the boy, who had previously been perfectly healthy, developed measles. During convalescence he had a series of convulsions followed by unconsciousness for one week. On recovery he was unable to sit up in bed without support, and showed the most marked ataxia of his legs. Three months later he was still very ataxic, but eight months later he had made some improvement; twenty months after the onset of his disease he showed very little abnormality, although his mother stated that his moral sense was still perverted.

The case recorded by Dr Frederick Taylor has especial interest, for it shows that complete and absolute recovery may take place, so that in adult life the patient is able to undertake and perform the usual duties of life. A boy aged 4 years was first seen in 1875. He had had whooping-cough three weeks previously. He had trembling of his limbs and trunk which closely resembled disseminated sclerosis. In 1878 he had considerably improved, but his speech was still imperfect. In 1904 he was 33 years old, and no sign of his former complaint existed. He was an active and busy man.

Under the third heading, progressive cerebellar ataxia, two cases are described in which ataxia, starting at the age of 9 and 13, slowly and steadily progressed. The close relationship of this group to Friedreich's disease and to hereditary ataxia is recognised, but the cases recorded differ in many particulars from these diseases.

The pathological evidence for regarding these cases as due to cerebellar lesions is then discussed. It is shown that changes in the cerebellum are the most constant feature in this affection, though such changes are frequently accompanied by other lesions of, or lack of development in, the cerebrum, pons, medulla, or spinal cord. It is known that almost complete atrophy of the cerebellum may exist without giving rise to any symptom, and in explanation of this fact the author quotes the work of Luciani, who showed that animals rapidly recover from symptoms produced by ablation of the cerebellum if the cerebral cortex remained undamaged; but if the cerebral cortex was injured in the region of the opposite gyrus sigmoidæ, then the symptoms produced by removal of the cerebellum persisted.

The diagnosis from disseminated sclerosis, quiescent cerebellar tumour, and hydrocephalus is discussed.

The prognosis with regard to the congenital cerebellar ataxia and the acute ataxia is relatively good, whereas the prognosis in the progressive cerebellar ataxia is bad, but some cases of this latter group run a very prolonged course.

AUTHOR'S ABSTRACT.

**CORTICAL TACTILE PARALYSIS.** (*Die kortikale Tactlähmung.*)  
(149) FRANZ KRAMER (Breslau), *Monatssch. f. Psych. u. Neurol.*, Feb. 1906, p. 129.

IN this paper the author goes into the question of the relationship between pure tactile sensation and the other forms of sensibility (particularly the sense of position and the sense of movement) with regard to the perception of tri-dimensional form. A number of cases are detailed in which there were disturbances from cortical lesions of tactile sensation with more or less affection of other sensory qualities. One case in particular he regards as a practically pure tactile paralysis—the sense of movement was slightly impaired, the perception of simple touch was perfect, and the power of localisation was intact, but there was complete failure of the power of recognising the shape and form of objects. Such loss of the stereognostic sense he considers due to a disturbance of cortical association, so that there is failure to combine the successive tactile pictures coming from the periphery into what becomes a sensation of form. In other cases the loss may be due to failure to combine the two dimensional stimuli coming from the skin with the sense of position of the perceiving fingers. But all such pure tactile paralyses must always be the result of a superficial cortical lesion—deeper affections cause in addition (*inter alia*) disturbances of other forms of sensibility.

J. H. HARVEY PIRIE.

**ON THE PRESENCE OF KERNIG'S SIGN IN HERPES ZOSTER.**

(150) (*Sur la présence du signe de Kernig dans le zona.*) RAYMOND BELBÈZE, *Arch. Gén. de Méd.*, Feb. 27, 1906, p. 520.

DURING the past four years the writer has seen nineteen cases of herpes zoster; and in examining them, has been on the outlook for any sign that would give clinical support to the hypothesis that the disease is of central nervous origin. Pathological evidence and theoretical considerations are in favour of this view of the etiology, and the arguments brought forward against it are of little weight. Of the nineteen cases recorded, two showed an absolutely definite Kernig's sign. Both patients were healthy women past middle life, and in both the eruption was on the abdomen. One of them at the onset of the disease was suffering from a very severe and intractable attack of constipation. It was impossible to be sure that Kernig's sign had not been present before the attack; but it was observed that it became gradually less marked as the eruption subsided, and disappeared entirely with recovery. The writer strongly opposes the theory advanced by M. Amaducci (*Il Policlinico*, 1905) that Kernig's sign is the result of a peripheral irritation acting on a normal centre, on the ground that were the centre in a normal condition it could not, when stimulated from a peripheral source, give rise to a pathological manifestation.

HENRY J. DUNBAR.

**BULBAR SYMPTOMS OCCURRING WITH CARCINOMA OF  
(151) PARTS OTHER THAN THE NERVOUS SYSTEM, AND  
RESULTING FROM INTOXICATION.** T. H. WEISENBURG,

*Univ. of Pennsylvania Medical Bulletin*, Jan. 1905.

CEREBRAL symptoms may occur in persons suffering from carcinoma of internal organs. Aphasia, convulsions, hemiplegia, or monoplegia are commoner than paralysis of cranial nerves and bulbar symptoms.

Indications of bulbar palsy are the least frequent. Invasion by microscopic metastases, especially in the pia mater, vascular changes, and toxic influences are the supposed causes. Weisenburg relates the case of a woman, æt. 59, who suffered from recurrent carcinoma mammæ. Five months before death difficulty in swallowing was noticed, and chromatolysis was found in the cells of the nucleus ambiguus and of the dorsal vagus nucleus, and a lesser alteration in the cells of the nuclei of the glossopharyngeal, facial, and abducens nerves. The author gives some review of the

literature. The abstractor described a comparable case in this journal, published after the appearance of Weisenburg's note.

W. B. WARRINGTON.

**SPINDLE-SHAPED ENLARGEMENT OF THE BLIND SPOT  
(152) ASSOCIATED WITH CONGESTION OF THE OPTIC DISC.**

A. MAITLAND RAMSAY and W. M. SUTHERLAND, *Ophthalmic Review*, Jan. 1906.

THE authors of this interesting and important paper, while using Bjerrum's screen in testing the state of the field of vision in a case of sympathetic ophthalmia, demonstrated the presence of a vertical spindle-shaped elongation of the blind spot associated with congestion of the disc. The condition of the blind spot was determined by the same method in five cases in which congestion of the disc was found with other evidences of sympathetic ophthalmia, and in each case vertical spindle-shaped elongation of the blind spot was found. Reference is made to the normal appearance of the blind spot as demonstrated by this method. The surrounding amblyopic zone, which in health shows irregular extensions at the upper and lower margins where the large vessels pass from the disc, is described. The figures given show how remarkable is the change in the shape of the blind spot.

In the normal the vertical diameter is to the horizontal as 6 to 4. In the cases of sympathetic ophthalmia examined, the horizontal diameter remains unchanged, but the vertical becomes much increased. In the five cases examined the relative increase of the vertical diameter is represented by the following figures: First case, 12 to 4; second, 12 to 4; third, 10 to 4; fourth, 12 to 4; and fifth, 18 to 4.

The opinion of the authors as to the clinical importance of this change in the blind spot may be gathered from the following passage in their paper: "If, then, in a case of infected wound, or of degenerative change in one eye, its fellow began to give trouble, no matter how slight, and careful examination showed a congestion of the disc with characteristic enlargement of the blind spot, the sign would in our opinion go far to determine the question of the immediate enucleation of the exciter.

The explanation offered for the appearance of this change in the shape of the blind spot is, that the congestion of the disc and turgescence of the large retinal vessels accentuate and increase the extensions of the amblyopic zone round the blind spot, which normally are met with at the upper and lower margins, and thus the spindle-shaped enlargement in the vertical diameter is produced.

A. H. H. SINCLAIR.

**CONGENITAL AND HEREDITARY BILATERAL OPHTHALMO-**  
**(153) PLEGIA EXTERNA.** (*Ophthalmoplégie externe bilatérale*  
*congénitale et héréditaire.*) J. CHAILLONS et P. PAGNIEZ,  
*Nouv. Icon. de la Salpêtr.*, nov.-déc. 1905, p. 666.

IN this paper, notes are given of four cases of ophthalmoplegia externa occurring in three generations of the same family. In the first and second generation, malformation of the anterior part of the cranium, and immobility of the frontal portion of the occipitofrontalis muscle, were found as concomitants of the ocular condition.

In all four cases the eyes occupied a position of divergence. The vertical movements were absent, and convergence was only observed to a very limited degree. Nystagmus was present in all, and in some corneal opacities were found. The pupil reaction was normal in all, and accommodation so far as could be ascertained was normal also.

The first patient had a good hereditary history, but three out of her seven children had ophthalmoplegia externa, and one of the three became the mother of a child also affected with the same condition.

The authors regard the lesions in the cases they describe as probably nuclear. They affirm that the results of several post-mortem examinations in similar cases give support to their view that a developmental defect in the oculo-motor centres is present in these cases.

A. H. H. SINCLAIR.

## PSYCHIATRY.

**A STUDY OF DEMENTIA PRÆCOX.** D'ORSAY HECHT (of Chicago),  
 (154) *Journ. of Nerv. and Ment. Dis.*, Nov. and Dec. 1905.

THE literature, from Heinroth (1818) to Kraepelin (1904), is briefly reviewed, for the purpose of pointing out the different phases in the evolution of this disease picture. Hecker in 1871 contributed his masterly work on "Hebephrenia," the chief features of which he correlated as follows:—"Onset in close succession to puberty; the appearance, alternately, of melancholy, maniacal and confused states; a speedy psychic decline, with its finality in terminal dementia, which may be anticipated from the first." Kahlbaum followed with his "Heboidophrenia," a curable type of "Hebephrenia," and later with his classical paper on "Katatonia." Tuke in 1879 referred to the psychic disturbances which developed at puberty, and led to progressive mental weakness. Clouston in

1888 insisted that the essential feature of many adolescent cases was the tendency toward dementia from the first. Hecht then enumerates in chronological order the contributors whose works have materially enriched the literature on this subject since 1883. The latest, the most original, and the most illuminating contribution to the study of this series of disease pictures has been furnished by Kraepelin. With one sweep Kraepelin has correlated certain maniacal states, depressed states with stupor and catalepsy, bizarre attitudes with delusions and hallucinations, to form one comprehensive group, "Dementia Præcox," whose termination is in a special form of mental reduction. This group corresponds to the Adolescent Insanity of the English, the Dementia Primitiva of the Italians, the Jugendirresein of the Germans, the Démence Précoce of the French, and the Primary Dementia of the Americans. The general symptomatology of Dementia Præcox with its clinical types is considered, and brief cases are introduced to illustrate each type. Pathological research has revealed little, but the changes noted have been suggestive enough to stimulate still keener investigation. The prognosis for recovery is better in the katatonic and hebephrenic than in the paranoid type of the disease. The existence of a paranoid type as a division of Dementia Præcox is contested by many. The differential diagnosis from circular insanity, paranoia, and general paralysis is sometimes difficult, because of the numerous and varied symptoms which this disease picture may present.

C. H. HOLMES.

**THE TIME OF SOME MENTAL PROCESSES IN THE RETARDATION AND EXCITEMENT OF INSANITY.** SHEPHERD IVORY FRANZ, *Am. Journ. of Psych.*, Jan. 1906.

FRANZ states that this work was undertaken in order to aid in the solution of the problem, to what parts of the nervous system can the decreased and increased psychomotor activity found in manic depressive insanity be referred? He reviews briefly the clinical symptoms of this form of insanity, and adds the histories of six subjects used in his experiments—two normal, two retarded, and two exhilarated subjects. The experiments deal chiefly with the time consumed in certain mental processes, and their object is to determine the amount of slowing in the retarded, and the increase in ability of the exhilarated subjects. Seven kinds of experiments were made: (1) the time of rapid tapping; (2) the time of simple reaction to sound; (3) the time of choice reactions to sound; (4) the rapidity of reading; (5) the time of discriminating and marking out letters; (6) the time of adding; (7) the time of discriminating and distributing coloured cards. Results are grouped by weeks,



and the weekly averages are collected into tables. The number of experiments varied from 5 a week in the tapping-time, to 100 a day in the reaction-time experiments. Franz concludes that the exhilarated patients do not show a consistent increase in speed over the normal or the depressed patients. Therefore the maniacal state is simply an increased motor diffusion, and not an increased motor ability. The retarded subjects were slow in the beginning, but the retardation was not regular throughout the series of experiments.

In the complex processes the retarded subjects took proportionately less time than they took for simpler acts. The exhilarated subjects kept the normal relations. The movements of the retarded subjects became more rapid after practice. Systematic exercise will not cure the depression, but it causes improvement by lessening the retardation. The habit of slowness may be replaced by an activity habit developed through exercise. It is probable that retardation does not come at the beginning of the movement. The supposition that retardation may be due to a general lowering of the irritability is unsatisfactory, because it has not been settled just where this lowered irritability is. C. H. HOLMES.

**ON SOME RELATIONS BETWEEN APHASIA AND MENTAL  
(156) DISEASE.** SYDNEY J. COLE, *Journ. of Ment. Sci.*, Jan. 1906,  
p. 28.

APPLYING to echolalia the dictum of Wernicke—that the speech-phenomena of mental disease can often be advantageously regarded from an aphasic standpoint—the author discusses the aphasic relations of this well-known psychiatric symptom. He shows in various ways its relation to failure of understanding for spoken language, and regards it as commonly indicative of an aphasic disorder approximating to the so-called “transcortical sensory aphasia” of Wernicke (Lichtheim’s “Type VI.”). Such a disorder, transient or permanent, may not seldom be observed as one of the expressions of an insanity. As a transient disturbance it is observed in acute intoxications (von Monakow), in post-epileptic conditions (Pick), in epileptic confusional states not obviously attributable to fits (Raecke), and in other forms of insanity of confusional type. The author relates a case of insanity in which it accompanied Jacksonian fits referable to irritation of the motor area for the right upper extremity. He also illustrates its occurrence in re-evolution after general paralytic seizures. The occurrence of transcortical sensory aphasia as a permanent condition is then discussed, with special reference to cases presenting no coarse focal lesion. Many of these are cases of senile dementia,

in which a general diffuse atrophy of the cerebrum has been most marked in the left temporal region. Abstracts are given of ten of the most important observations (Pick, Liepmann, Heilbronner, and others). A similar aphasic disorder, often of slow and insidious onset, occurs also in dementia following insanity of comparatively early life. Cases of this class, in part assignable to dementia præcox, have as yet been little studied, and do not appear to have been in any instance reported in detail; the author accordingly gives a full analysis (6 pp.) of a case of presumable katatonic dementia præcox, beginning at the age of 20. The patient, whose age at the time of writing was 63, had for many years exhibited a condition nearly resembling transcortical sensory aphasia. Marked automatic echolalia was the most conspicuous feature. There being nothing suggestive of coarse lesion, the author suspects a diffuse atrophy predominating in the region of the left temporal lobe. He reviews the various explanations advanced for the production of echolalia, and inclines to that of Pick, based upon Hughlings Jackson's doctrine of inhibition. Certain phenomena of reading, associated with echolalia, are interpreted in similar fashion. A bibliography is appended.

AUTHOR'S ABSTRACT.

**ON THE DISCLOSURE OF FACT BY PSYCHOLOGICAL METHODS.**

(157) (*Zur psychologischen Tatbestandsdiagnostik.*) C. G. JUNG (of Zurich), *Centralbl. f. Nervenheilk. u. Psych.*, Nov. 1, 1905.

THE author gives here an example of the practical application of his association method to criminal psychology. If by association-tests submerged complexes with unpleasant effect could be diagnosed, it seemed possible by this means to demonstrate the presence of a complex of ideas related to a crime committed. Jung applied his tests to a boy suspected of theft, and by means of the reactions to special words inserted in the series was able to convince himself of the guilt of the patient, and thus extract a full confession.

C. MACFIE CAMPBELL.

**EXPERIMENTAL OBSERVATIONS ON MEMORY. (Experimentelle**

(158) *Beobachtungen über das Erinnerungsvermögen.*) C. G. JUNG (of Zurich), *Centralbl. f. Nervenheilk. u. Psych.*, Sept. 1, 1905.

IN association experiments with hysterical cases, the author frequently observed that, when the test word given to the patient touched upon the dissociated complex of ideas at the bottom of

the disorder, the patient frequently failed to react, and then after some time asked what the given word was. It was demonstrated that patient had really forgotten the word, this being simply one instance of the general tendency to submerge an unpleasant complex.

It is important in treating such cases to learn the content of the submerged complex; one indicator of a complex is the long reaction-time when the word given refers to the complex. In this article, Jung calls attention to the fact that when one asks the patient, after the series of associations has been completed, to give again the same reactions, the patient makes several mistakes or has forgotten the reaction. He found that the associations, where the memory of the patient was at fault, were exactly those relating to the complex. He gives two cases with complete series of associations to demonstrate this point. The influence of feeling-tone on memory is clearly demonstrated; an unpleasant effect leads to increase in reaction-time and to forgetfulness of the reaction. Wherever in mental life there is a submerged complex, we meet the same phenomenon but in different degrees—the transitory embarrassment of the normal individual, the “voluntary” amnesia of the hysterical, and the blocking in the catatonic.

C. MACFIE CAMPBELL.

**APRAXIA IN GENERAL PARALYSIS.** (*Apraxie bei progressiver (159) Paralyse.*) M. LEWANDOWSKY (of Berlin), *Centralbl. f. Nervenhe. u. Psych.*, Sept. 15, 1905.

PATIENT was a day-labourer, aged 36, who had suffered from epileptiform attacks. The psychosis developed in a manner which suggested at first the diagnosis of catatonia; there were periods of excitement with hallucinations.

When observed by the author, patient had complete motor aphasia, contracture of the right arm; patient seemed unable of his own accord to sit up, raise himself from his chair, walk backwards or forwards. The left arm showed no paralysis, but only was used in three different movements; these were, to put his hand behind his ear as if to hear better, to move his hand to his mouth, and to rub his head with his left hand. The essence of the disorder seemed to be of the nature of a motor apraxia, and not to be sufficiently explained by the dementia or by agnosia. In addition, patient presented choked disc with fresh hæmorrhages. Post-mortem examination showed a typical general paralysis.

C. MACFIE CAMPBELL.

**GENERAL PARALYSIS AND TRAUMA. (Paralyse und Trauma.)**(160) C. GIESELER (of Königsberg), *Arch. f. Psych.*, Bd. 40, H. 3.

AFTER reviewing the opinions of others on the relation between general paralysis and trauma, Gieseler gives six cases in which the two were associated (one case with psychical trauma). In only two of these was the trauma an ætiological factor, in the other cases the association being casual, or the trauma due to the already existing paralysis. As to the exact rôle of the trauma he is cautious, and says that it is improbable that trauma *per se* can be a sufficient cause of general paralysis.

C. MACFIE CAMPBELL.

**THE PUPIL ACCOMMODATION REFLEX IN GENERAL**(161) **PARALYSIS. (L'accommodateur dans la paralysie générale.)**MARANDON DE MONTYEL, *Journ. de Neurol.*, Feb. 5, 1906, p. 41.

FROM prolonged and minutely detailed observations in the cases of 140 general paralytics, the author draws the following conclusions :—

1. In any case of general paralysis which has passed through all the stages of the disease, some abnormality is to be found at one time or another in the pupil accommodation reflex.

2. In the great majority of cases accommodation in both eyes is simultaneously and similarly affected, the alteration generally being in the direction of enfeeblement or abolition.

3. In the first two stages of the disease the tendency is to enfeeblement rather than to abolition ; in the third stage abolition is the more frequent.

4. In the same patient it is most common to find abolition following enfeeblement. In rarer cases, when no change occurs in the abnormality, enfeeblement is twice as common as abolition. In exceptional cases, exaggeration, enfeeblement, and abolition may occur in the same patient.

5. In the first stage accommodation is abnormal in 50 per cent. of cases ; in the second stage in 66 per cent. It is exceptional to find it normal in the third stage, but it may be so.

6. Exaggeration is met with only in the first stage. Different conditions in each eye occur only in the first and second stage. In the last stage only enfeeblement and abolition are found, and the changes are identical in both eyes.

7. Abolition of accommodation, in all probability, is never produced as a primary condition, but is preceded by enfeeblement.

8. The two changes of enfeeblement and abolition are in direct ratio to the progress of the disease, but enfeeblement is more common in the initial stages and abolition in the terminal.

T. C. MACKENZIE.

## Review

**HISTOLOGICAL STUDIES ON THE LOCALISATION OF CEREBRAL FUNCTION.** ALFRED W. CAMPBELL, M.D., xix., 360 pp. roy. 4to, 29 Plates, Cambridge, 1905.

It would be difficult, we think, to speak too highly of this most important and valuable research. It is a monumental work, which reflects the greatest credit on Dr Campbell and on British medicine; it should be carefully studied by everyone who is interested in the anatomy, physiology, and pathology of the brain, and in the clinical study of neurology.

In his preface the author states "that a study of the cortex cerebri in the normal state constitutes the basis of the present research, and may be regarded as the corner-stone in the histological foundation upon which the superstructure of cerebral localisation may be reared by workers in other departments." He goes on to say: "that with rare exceptions previous observations on the structure of the cerebral cortex have been founded on what may be termed piecemeal work, and that it is plain that observers have previously baulked an attempt to explore the whole surface in a comprehensive and complete manner on account of the magnitude of the task." He now claims to have accomplished this undertaking. He has made a collateral comparison of the cell lamination and fibre arrangement in section after section, and millimetre by millimetre, over the entire surface of the human cerebrum. In more than one case he has converted an entire cerebral hemisphere into serial sections, and has alternately stained them for the display of nerve cells and nerve fibres.

The *material* used is divisible into three categories, namely, normal human, normal comparative, and pathological.

The normal human material consisted of: three cerebral hemispheres completely examined for both nerve cells and nerve fibres; three hemispheres completely examined for fibres only; and two hemispheres partially examined for nerve cells and nerve fibres.

Six of the above mentioned hemispheres were taken from persons who died while of unsound mind in Rainhill Asylum. In anticipating criticism on this point, Dr Campbell makes the following important statement:—

"It may be urged that the mere fact of a person having suffered from insanity is sufficient in itself to condemn the brain as unsuitable material for an investigation of this description. In reply to this, while I confess on looking back that I should have preferred that more brains in the series were

from individuals free from mental disorder, it is almost needless for me to say that I should not have continued to employ the insane brain had I not felt that the objections to its use were based more on sentiment than reality, and had I not convinced myself from a lengthy experience in the pathological laboratory attached to Rainhill Asylum that, in a large proportion of cases dying insane, all the microscopic methods at our disposal will fail to disclose changes, either in the nerve cells or fibres, which we can refer to their altered mental condition; and that in other cases in which the mental disorder is more advanced or of a graver nature, while we may be able to discover alterations in the nerve cells—thanks to the marked advance which has been made of late years in this province of histology—yet the present state of our knowledge will not allow us to make any definite declaration concerning attendant changes in the nerve fibres. And I am able to speak without reservation of the difficulties which beset the detection of morbid changes in the nerve fibres in the cerebral cortex, because for several years prior to the inception of the present investigation I devoted much time to a study of this subject.”

The normal comparative material included: (1) The right hemisphere of a Chimpanzee, completely examined for nerve fibres and partially investigated for nerve cells. (2) The left hemisphere of a Chimpanzee (another animal), completely examined for fibres only. (3) The right hemisphere of an Orang, completely examined for fibres only.

The pathological material consisted of:—two brains from cases of amyotrophic lateral sclerosis, seven from cases of amputation of one or other extremity, three from cases of tabes dorsalis, and one from a case of old-standing capsular lesion—in all of these the central convolutions and parietal lobe were examined—and two cases of old-standing blindness, in which the occipital lobe was completely examined.

In the Addendum, the histological characters of the cortex of the brain in the cat, the dog, and the pig are described, and the functions and homologies of the more important cortical areas are considered in considerable detail.

With regard to the *methods of examination*, all the normal human and the anthropoid brains were hardened in Müller's fluid or in Orth's solution—a mixture of Müller's fluid and formalin; after fixation, orthogonal tracings were drawn, showing the exact disposition of the convolutions and sulci on the various surfaces, and to confirm the correctness of these tracings the same surfaces were photographed. In the case of the anthropoid brains a plaster of Paris cast was always made, and proved of great assistance in facilitating orientation when the preparations were ready for microscopic examination.

The hemispheres which were completely examined were first divided into portions of suitable size for section on the microtome. The lines of cleavage between the blocks were carefully and correctly indicated on the original tracings and photographs. Then the blocks were numbered and placed in separate bottles

after hardening in increased strengths of alcohol and imbedded and cut in celloidin on a Jung microtome. The sections, of a thickness of 25  $\mu$ , were taken at intervals of 1 mm. and preserved in strict serial order between sheets of paper and subsequently mounted and stained; and so, in the case of the central convolutions, for instance, sections were obtained showing their structure at about a hundred different levels.

For staining the nerve fibres the method known as that of Wolters-Kulschitzky was adhered to throughout. The nerve cells were stained by a  $\frac{1}{4}$  per cent. solution of thionin. By this method, the author states that "in spite of the thickness of the sections he was able to see the Nissl bodies clearly, and, what was more important for his purpose, the cell morphology and lamination were shown to perfection." He adds: "I cannot too strongly emphasise the advantage in time-saving and general convenience secured by the possibility of staining first cells and then fibres in successive series of large sections, for apart from the obvious advantage in obtaining a ready comparison between cell and fibre constituents in given parts, it converts a comprehensive investigation of cortical cell lamination from a gigantic and almost impossible task, when small sections are employed, into one of easy accomplishment."

The enormous labour involved in this research is shown by the fact (which the author states in a footnote) that the examination of a whole human hemisphere by this method takes *six months* for its accomplishment (whereas to go over it thoroughly in small blocks would absorb at least two years). When it is remembered that Dr Campbell has examined no less than three human cerebral hemispheres completely for both nerve cells and nerve fibres, three hemispheres completely for fibres only, and two hemispheres partially for nerve cells and nerve fibres, and in addition a large amount of normal comparative and pathological material (enumerated above), the extraordinary magnitude of the work on which Dr Campbell's splendid research is based will be readily appreciated.

The work consists of eleven chapters and an Addendum.

Chapter I. deals with the material and methods of examination; Chapter II. with general remarks on cell lamination and the arrangement of the nerve fibres in the cerebral cortex; Chapter III. with the precentral or motor area; Chapter IV. with the post-central or sensory area and the intermediate post-central area; Chapter V. with the visuo-sensory and visuo-psychical areas; Chapter VI. with the temporal lobe and the auditory areas; Chapter VII. with the limbic lobe; Chapter VIII. with the parietal area; Chapter IX. with the intermediate precentral area; Chapter X. with the frontal and prefrontal areas; and Chapter XI.

with the island of Reil. The Addendum includes "further histological studies on the localisation of cerebral function; the brains of felis, canis, and sus compared with homo."

Each of the chapters devoted to individual parts of the cerebral cortex comprises: (1) a detailed description of the cell lamination and fibre arrangements of the special part under investigation; (2) a discussion on the functions of the part examined, based upon the histological characters of the cortex, the results of experimental investigation, clinico-pathological data, the teaching of comparative anatomy, and developmental evidence; (3) a summary of the author's histological study of the part of the cortex under discussion and of his conclusions as regards its function (based on a consideration of the various data enumerated above); and (4) a very complete list of references and authorities.

The written description of the histological characters of the different parts of the human cerebral cortex is illustrated by twenty-three figures in the text and twenty-five full-sized plates and legends, and the Addendum is illustrated by four full-sized plates.

Many of the microscopic drawings of the preparations were made by Dr A. C. Wilson and are a most important and valuable feature of the work, showing as they do with the greatest accuracy and faithfulness the exact cell lamination and fibre structure in individual parts of the cerebral cortex of man; for the purpose of reference and comparison, these drawings will be of the greatest value to future workers. Most of the photographic work incidental to the research was done by Mr F. J. Abram.

The microscopic drawings were made by means of the eye-piece drawing apparatus of Leitz. Dr Campbell states—and on this point we entirely agree with him—that perfectly accurate drawings made in this way are superior to microphotographs. He claims that "in the low power drawings the position of every fibre, at any rate in the outer two-thirds of the cortex, is faithfully shown, and that in the high power figures the calibre as well as the relation of the fibres one to another is accurately represented, and that as regards the cells, size, position, and number are faithfully shown." All the drawings were made to scale, the low power ones at a magnification of  $\frac{80}{1}$ , the high power at  $\frac{480}{1}$ .

From what has been stated, it will be seen that Dr Campbell has described in great and exact detail the histological characters of the whole cerebral cortex in man, and has figured his results in a series of most beautiful and accurate microscopic drawings. Further, the gross results of his research are diagrammatically represented (so far as it is possible to represent them in a surface view) in several charts (maps and legends), which are of great interest and value.



So much as regards the histological observations and matters of fact.

With regard to the *conclusions*, as to the physiological functions of the different areas, which Dr Campbell draws from his histological studies and from other data (experimental, clinico-pathological, embryological, comparative, etc.), there is naturally much room for difference of opinion; many of the author's conclusions must in the meantime be regarded as suggestions—suggestions of great value—which will have to be proved or disproved by further observation. In connection with this part of the work, Dr Campbell shows a wide acquaintance with the work of previous observers, an extensive literary knowledge, a keen critical faculty, and a discriminating judgment. The reading involved and the consideration required to form a judgment on the numerous points considered with regard to function must have been very great.

In the limited space of a review it is impossible to describe in detail Dr Campbell's histological findings or to discuss and criticise the many debatable points involved in his conclusions; but it may be useful to indicate some of the facts and conclusions which have a direct bearing upon clinical medicine.

*Chapter III. Precentral or motor area.*—The author confirms the important conclusion which Sherrington and Grünbaum came to as the result of electrical stimulation of the brain of the anthropoid apes, viz. that the motor area is practically confined to the precentral (ascending frontal) convolution. Both in the anthropoid apes and in man, Dr Campbell has been able to map out a histological area which agrees very closely with that which responds to electrical irritation. The floor (not the lip) of the fissure of Rolando forms a very definite and constant posterior limit to this area.

The same area corresponds approximately to the distribution of the giant or "motor" cells of Betz and Bevan Lewis. The giant cells disappear before the lower extremity of the fissure of Rolando is reached, and are consequently not found over that part of the cortex which is regarded as the face area; in this area, however, large cells are found which differ from the large pyramidal cells common to the whole precentral area; they are possibly special presiding elements.

Strong confirmation of the assumption that in man as well as in the anthropoid apes the motor area is confined to the precentral gyrus and its paracentral annex is afforded by the condition of the ascending frontal and ascending parietal convolution in amyotrophic sclerosis, and in cases of amputation of the limbs.

In two cases of amyotrophic lateral sclerosis (a disease in which the lesion is limited to the muscular system and the motor system of neurones) there was a wholesale disappearance of the "motor"

cells throughout the normal area of their occupation, and while there was a co-existent disturbance of other elements in the pre-central cortex, the post-central gyrus entirely escaped affection.

With regard to the amputation cases, Dr Campbell states that in the seven cases which he has examined he has never failed to find microscopic changes, akin to, if not absolutely identical with, those to which Marinesco has given the name "*réaction à distance*." He maintains that "given a case of section of the nerves supplying even a single group of muscles, for instance, the extensors of the foot, it would be quite possible from a careful examination of the cortex and a study of the resulting "*réaction à distance*" to determine the exact distribution of the motor elements, on the integrity of which, movements of that particular group of muscles depended, and, by collecting and examining a selected series of similar cases and collating the results with the findings of the physiologist, the clinician, and embryologist, we may eventually hope to draw on the surface of the human brain a detailed map of motor localisation, so definite and so exact, that it will not require alteration and revision at the hands of our successors." The exact alterations found in the seven amputation cases which he examined were as follows:—

"In two cases of amputation of the leg a short distance below the knee, I have found," he says, "changes limited to the upper extremity of the precentral gyrus and its paracentral annex, in other words to the part which in the case of the higher ape seems to control movements of the toes and ankle. In another case of amputation at the knee-joint, associated with great atrophy of the thigh muscles, the changes extended further outwards, but numerous cells above the superior annectant gyrus remained intact; the latter probably govern hip movements. In two cases of amputation of the arm through the humerus, degenerated cells were found over an extended area corresponding very closely with Professors Sherrington and Grünbaum's experimentally located areas for finger, wrist, and elbow movements; and in one of these cases, which was associated with extreme wasting of the shoulder muscles, a large group of cells lying immediately below the superior annectant gyrus was affected. In a case of amputation of the hand the changes were limited to the lowermost part of the last-mentioned area."

Dr Campbell states that it is impossible to reconcile these findings with the long list of clinical observations adduced in the past to support the view that the two central convolutions have an equal share in the control of volitional movements, and it is suggested that natural lesions such as cerebral softening, cerebral tumour, and cerebral trauma, which form the basis of most of these observations, are only in rare instances sufficiently limited in their effects to allow of safe judgment on this question; hence errors have arisen.

He states that the conclusions deduced from clinical observations, from experimentation, and from histological investigation,

are completely in agreement concerning the sequence of representation of movement along the course of the motor area.

Dr Campbell argues that the precentral (ascending frontal) convolution is a purely motor area; he does not agree with those (Bastian, Munk, and Mott) who think that it is the seat (or a seat) of the muscular sense, or with those (like the late Dr Ross) who think that it is a combined sensory and motor area.

*Chapter IV. Post-central or sensory area.*—The author states that "structurally the post-central (ascending parietal) gyrus differs entirely from the precentral (ascending frontal) gyrus and from the superior parietal and supramarginal convolutions, and its definition as a distinctive area is accomplished without the slightest difficulty. It is not nearly so rich in nerve fibres as the precentral gyrus, and a most important distinguishing feature is the presence in the inter-radiary plexus of fibres of even larger calibre than those seen in the precentral convolution, fibres which are curious, inasmuch as they run obliquely or at right angles to the radiating fasciculi. Such fibres recall some met with in known sensory regions, for instance, the visual, auditory, and olfactory centres. They are not seen in either the precentral convolution or the parietal lobe proper, they seem to concentrate themselves on the Rolandic side of the gyrus, and their curious oblique course gives rise to the assumption that they are centripetal fibres making for cells resident in this situation."

In reference to nerve cells, the lamination differs from that of the precentral gyrus, first, in showing no true cells of Betz; and, secondly, in exhibiting a most pronounced layer of stellate cells; and it differs from that of the remaining parietal region in containing pyramidal cells of larger dimensions.

The largest of these pyramidal cells are smaller than an average-sized precentral giant (motor) cell; they are pyramidal in shape, while the Betz cells are pyriform; their apical extension process tapers away more gradually; unlike the typical cells of Betz, these elements are not found lying in nests or clusters, but are solitary.

That these large pyramidal cells in the post-central gyrus are sensory is proved by the facts that in amyotrophic lateral sclerosis (in which the Betz cells are destroyed and atrophied) they are unaffected, and that in tabes dorsalis (in which the Betz cells are unaffected) they are profoundly affected (degenerated and atrophied).

Dr Campbell argues that this post-central gyrus constitutes the terminus where the main system of fibres for the conveyance of impressions pertaining to tactile and allied forms of sensation primarily impinges.

"The separate localisation of the various components combining to produce 'common sensation' is," the author states, "beset

with difficulties. However, the view is promulgated here that the post-central area, like better-known sensory realms, is divisible into a purely sensory part, to which all impressions primarily pass, and an investing psychic part. The former occupies the post-central area proper and, in accordance with my thesis, its destruction should lead to abolition of psychic, as well as impairment of fundamental sensory components; the latter covers the intermediate post-central field and may extend further back in the parietal direction; its destruction should lead to isolated disturbance of psychic sensory attributes. Some clinical and pathologic findings substantiate this view. The fact that fundamental attributes, such as the simple recognition of touch, pain, heat, and cold, are only dulled and rarely or never abolished in cases of cortical lesions, is probably due to the participation of subcortical intermediate stations in the receptive act."

In three cases of *tabes dorsalis* an examination of the brain disclosed changes limited to the post-central gyrus, very similar in character to those which he has described as the result of old-standing lesions in the internal capsule. The discovery of these changes Dr Campbell regards as of the greatest possible significance and one of the strongest points which can be advanced in favour of the view which he advocates, viz. that the ascending parietal convolution constitutes the terminus where the main system of fibres for the conveyance of impressions pertaining to tactile and allied forms of sensation primarily impinges. This point is of so much importance that we quote the paragraphs relating to it in full.

"I have now examined the cortex cerebri in three cases of *tabes dorsalis* in somewhat close detail, and in all three I have discovered important changes, almost gross in character. Having hitherto escaped notice at the hands of other observers, these changes, in themselves, constitute an interesting addition to our knowledge of the pathology of this disease, and would prove a fruitful topic of discussion in that light. Here, however, I am only concerned with cerebral localisation, and to this my comments must be restricted. Now, to my mind, the evidence derived from a study of these cases may be confidently advanced as stronger than any which has yet been adduced in favour of the assumption that the cortex of the post-central gyrus, and it alone, is the primary terminus or arrival platform for nerve fibres conveying impulses having to do with 'common sensation'; the data are so clear that they speak for themselves and need little in the way of introduction. For, just as we saw, in the last chapter, that amyotrophic lateral sclerosis, a disease confined exclusively to the muscles and the motor system of neurones, provided a convincing demonstration to the effect that the resulting cortical changes are limited in their distribution to what we may in the future call the 'precentral or motor area'; so we see in *tabes dorsalis*, a disease which is essentially a sensory one, and in typical cases exclusively confined to the sensory system of neurones, just as sharp a limitation of the associated cortical changes to the opposite bank of the Rolandic fissure, to what we may now designate the 'post-central or sensory area.' And, conscious as I am that the histological findings on which this weighty statement rests will need to be carefully

checked and confirmed by others before it can be considered final, I give it in the firm belief that the portrait received from the microscope is a correct one, and that the solution of a vexatious problem, which has baffled the neurologist for a number of years, is at hand.

"The limitation of the alteration to the post-central gyrus is the feature of predominant interest, but there are several points of minor importance which arise for consideration in this discussion. The first is that the alterations are still further limited to a certain part of the post-central gyrus, to the Rolandic wall and lip, to the field which was mapped out long before this pathological investigation was thought of, by its possession of a very curious and distinctive structure, the field which I have sometimes distinguished by the name post-central area proper."

*Chapter V. The visuo-sensory or calcarine and the visuo-psychic areas.*—Dr Campbell confirms the opinion of previous observers that the area of cortex characterised by the possession of the lamina or "line of Gennari" is the chief end station of the optic radiations, and therefore constitutes the cortical centre for the primary perception of visual sensations. The arrangement of nerve fibres in this area is absolutely distinctive, and it is also characterised by a special type of nerve cell lamination. "Briefly put, the characters which distinguish the calcarine type of cell lamination are, first, the almost unique external layer of large stellate cells usurping the position occupied by the external layer of large pyramidal cells in other regions; secondly, the existence of pale-stained zones above and below the uncommonly well-marked layer of stellate cells, the upper of which marks the position of the line of Gennari; thirdly, the presence in the depths of the cortex of the layer of solitary cells of Meynert, cells which differ from homonymous cells in any other part of the brain."

The distribution of this field of cortex (bearing a line of Gennari) is influenced directly by the calcarine fissure and follows closely every bend and branch of that sulcus.

The occipital or visuo-psychic type of cortex has decided and distinctive characters in the central parts of the field, but its exact distribution is not accomplished without difficulty. It may be described as a zone of cortex, from 1.3 to 2 cm. broad, investing this visuo-sensory area on all sides, that part above the stem of the calcarine fissure excepted.

In summing up his conclusions on the function of the visuo-sensory and visuo-psychic areas, Dr Campbell states: "Convinced from my histological investigations that two definite and distinct areas, each bearing a special type of cortex, can be mapped out in the occipital lobe, I am now satisfied, after a consideration of most of the recorded work on this subject, that these two fields have different physiological functions to perform. And joining hands with those who hold the belief that in the occipital lobe there exist two distinct cortical centres, one specialised for the primary

reception of visual sensations, the other constituted for the final elaboration and interpretation of these sensations, I would go a step further and affirm that the area of cortex in the calcarine region, which I have mapped out and termed visuo-sensory, represents the exact limits of the first-mentioned centre, while the investing field, which I have designated visuo-psychic, represents the precise extent of the second centre."

In this chapter the important subjects of psychic blindness, alexia, colour blindness, and optic aphasia are referred to.

*Chapter VI. The temporal lobe and auditory areas.*—The author maps out three distinct histological types of cortex in the temporal lobe. *Type 1* is confined in a remarkable manner to the transverse temporal gyri or gyri of Heschl. The leading features of this type of cortex are the presence in the radiary zone of numerous large fibres, the existence of a line of Kaes, and the general wealth of fibres in all layers; and, so far as the cell lamination is concerned, (a) the general rich supply of cells, and (b) the presence of numbers of curious giant cells above the well-developed stellate layer. *Type 2.* This forms a broad skirt or margin to the concealed area above mentioned (transverse gyri of Heschl), and is almost entirely confined to the first temporal lobe. Dr Campbell thinks that some part of this cortex spreads on to the insula and covers parts of the gyrus longus and gyrus posterior secundus. *Type 3.* This type covers a very large area and is contiguous with the "parietal" field, the dividing line between the two corresponding approximately but not absolutely with the disposition of the horizontal and occipital rami of the intraparietal fissure.

Dr Campbell states that the angular gyrus, which is supposed to possess special functions, does not differ structurally from other parts of the area. This is a point of special interest.

As regards function, Dr Campbell argues that the area of Type 1 (the transverse temporal gyri of Heschl) probably stands in relation to the auditory function in the same way as the calcarine region does to the visual, and is accordingly of prime importance as a centre for the primary reception of simple auditory stimuli.

He suggests that the only way of explaining the negative results which Schäfer observed after removal of the superior temporal gyrus is by supposing that his ablation of the superior temporal gyrus was not quite so complete as he imagined, and that he left behind part of the transverse temporal gyri of Heschl.

The area of Type 2 he regards as a second centre specialised for the interpretation and further elaboration of primary auditory stimuli. Dr Campbell could detect no difference in the structure of the corresponding areas on the two sides of the brain. "On the question," he says, "of a word-hearing" (does Dr Campbell mean

word-seeing) "centre in the left angular gyrus, histology affords negative evidence; for although I have subjected both hemispheres to examination I have been unable to detect any appreciable difference in the two sides, either in regard to the arrangement of nerve fibres or nerve cells."

In this chapter, deafness due to cortical lesions, word-deafness, amusia, and psychic deafness are considered in some detail.

*Chapter VII. The limbic lobe and olfactory area.*—The parts studied in this section include all the constituents of Broca's "grande lobe limbique," viz. the olfactory lobe (excluding the olfactory bulb and peduncle), the whole gyrus hippocampi (including the cornu ammonis and subiculum, the uncus and lobus pyriformis), the entire gyrus fornicatus, and other subsidiary structures. The author states that histology supports comparative anatomy in suggesting that, in the human brain, the lobus pyriformis must be regarded as the principal cortical centre, although not the sole one, governing the olfactory sense. Structurally the cortex of this lobe is not built up on the usual plan, and its chief distinguishing features are: (a) curious clusters or nests of giant polymorphous cells which occupy a unique position close beneath the surface; (b) a deep succeeding layer of pyramidal cells approximately equal to one another in size (S. Ramón y Cajal's tassel cells); (c) a correspondingly peculiar arrangement of cortical nerve fibres, of which the presence of projection bundles reaching right up to the zonal layer constitutes a prominent feature.

Dr Campbell does not agree with the view expressed by Ferrier and Horsley and Schäfer that common sensation is centred in the hippocampal region.

*Chapter VIII. The parietal area.*—The author applies the term "parietal" to an area which may be briefly described as covering the precuneus, the superior parietal gyrus, and the anterior part of the supramarginal gyrus. Structurally its cortex possesses all the cell laminæ of, and a similar arrangement of nerve fibres to, the "intermediate post-central" area, but it differs in containing a smaller number of special large pyramidal cells and of large medullated nerve fibres; it is also peculiar in showing a more perfect reduplication of the line of Baillarger.

To electrical excitation the area is irresponsive, and histology seems to favour the clinical doctrine that it shares with the "intermediate post-central" cortex the function of elaborating complex impressions embodied in the muscular and stereognostic senses.

*Chapter IX. The intermediate precentral area.*—The cortical field, which Dr Campbell terms the "intermediate precentral" area, ranges as a zone between 3.5 and 1 cm. in width, placed after the manner of a buffer in front of the "precentral" area proper and showing

an additional extension downwards on to the orbital surface of the hemisphere. Broadest above, the area becomes constricted at its middle and then expands again below. It covers the base of the upper and middle frontal gyri, some of the ascending frontal (that not coated by the "precentral" type), a considerable portion of the inferior frontal, including the pars basilaris (area of Broca), the pars triangularis (sometimes), and the pars orbitalis of the frontal operculum.

Histologically many of the structural characters noted in the "precentral" cortex are repeated; thus, the general depth is preserved, the difference in regard to nerve fibres chiefly affects the degree of fibre wealth, and save for the giant cells of Betz, the cell lamination is remarkably alike. These resemblances suggest a physiological kinship between the two parts. "Having," he says, "regard to the discoveries (1) that this cortex bears a structural resemblance as well as a topographic relation to the 'precentral' cortex, (2) that the field corresponds in distribution with the area found excitable in the simian brain by experimenters prior to Sherrington and Grünbaum, and (3), that its anterior boundary agrees to a marked extent with the so-called 'sensory projection centre' worked out by Flechsig on developmental lines, the proposition is favoured that it participates in the motor function; and it is submitted that it may represent a higher centre presiding over elements in the 'precentral area' proper, in short, that it is designed for the execution of skilled, as opposed to crude and automatic movements."

In this chapter the subjects of motor aphasia and agraphia are referred to. In connection with motor aphasia, Dr Campbell states:—

"Digressing to consider the localisation of the motor speech centre, it is submitted that this is probably not so restricted as previously supposed, and that the forward extension of the "intermediate precentral" cortex on the inferior frontal gyrus may have the same function as the cortex of Broca's area. In support of this assumption it is pointed out, in the first place, that histologically the cortex of all this part of the "intermediate precentral" area is alike, that is to say, the area of Broca is not distinguishable by any localised specialisation of structure; and, secondly, that it is a common matter of clinical experience that a superficial lesion confined to the cortex of Broca's area is not wholly effective in the production of motor aphasia; in other words, if the disability is to be permanent, the lesion must be deep and penetrating. The explanation given for the occurrence of complete and permanent motor aphasia after a deep-seated lesion in the pars basilaris is that all connections between the 'intermediate' cortex and the direct labial, lingual, and laryngeal centres occupying the lower end of the precentral area proper—and by the way remaining intact—are severed. Such a lesion therefore produces an effect equivalent to destruction of the whole of the 'intermediate precentral' cortex coating the inferior frontal gyrus."

The author adds that from this and from what we know regarding motor aphasia it is inferred that the "intermediate



precentral" cortex harbours a sequence of centres for the control of skilled movements, following the same order, deposited more or less on the same horizontal level, and connected by commissural fibres with the series of "primary" centres existing in the "precentral" area.

*Chapter X. Frontal and prefrontal areas.*—The part of the frontal lobe uncovered by "intermediate precentral" and "limbic" cortex comprises the anterior half of the marginal gyrus, on the mesial surface of the hemisphere, much of the superior, middle, and inferior frontal convolutions, on the lateral surface, and their downward extensions on the orbital face. Dr Campbell states that, although this expanse is covered all over by cortex showing a type of fibre arrangement and cell lamination approximately uniform in character, it is nevertheless possible to split it up into two fields, the hinder of which forms a skirt to the "intermediate precentral" area and will for convenience be called "frontal"; while the anterior, centred on the tip of the frontal lobe, will be designated "prefrontal."

The structural development of the "prefrontal" cortex is exceedingly low. It presents an extreme of fibre poverty; all its fibre elements are of delicate calibre, and its association system is particularly deficient. Its cell representation is on a similar scale. The cortex is also shallow.

The relative paucity of fibres and of nerve cells in the prefrontal lobe which Dr Campbell shows is very remarkable. It will be interesting to see whether his observations on this point are confirmed by subsequent observers.

The feeble structural representation of the "prefrontal" cortex suggests that it is the last portion of the frontal lobe to make its appearance in the course of phylogeny, and all things considered, the idea is favoured that its physiological importance as a psychic centre is over-estimated; the same does not apply to the "frontal" area.

*Chapter XI. The island of Reil.*—The general fibre supply of the insular cortex is poor, and it likewise contains no cells of large size. In the adult it is histologically separable into two main regions, an anterior and a posterior, between which the sulcus centralis insulæ roughly forms a dividing line.

Dr Campbell thinks that "it is probable that the insula is 'old' in the rank of phylogenesis, and that it plays a more important part in primitive mammals than in man and the higher apes."

"Histology," he says, "does not support the view that the insula is endowed with speech functions. In the cases giving rise to this conception, an extension of the destroying lesion, either to the inferior frontal gyrus or to the transverse temporal gyri, is

suspected. Clothed by temporal cortex, the posterior insula is supposed to pertain to the auditory apparatus. Studies in comparative anatomy show a close topographic relation between the insula and the fissura rhinica, and as the anterior insula contains elements common to the olfactory area, it may have to do with the recognition of smell; or in accordance with Gorschkow's experiments, its specialised cortex may represent the gustatory centre."

In conclusion, we beg to congratulate the author most heartily on this most laborious and splendid piece of work. We repeat that it reflects the greatest credit on British medicine, and that it should be most carefully and thoroughly studied by everyone who is interested in the structure and functions of the brain and in the clinical study of neurology.

The book is excellently printed and got up, in particular the admirable way in which the microscopic drawings have been reproduced deserves to be mentioned.

BYROM BRAMWELL.

# Bibliography

## ANATOMY

- ECONOMO. Beiträge zur normalen Anatomie der Ganglienzelle. *Arch. f. Psychiat.*, Bd. 41, H. 1, 1906, p. 158.
- SJÖVALE. Über Spinalanglienzellen und Markscheiden. *Anatom. Hefte*, Bd. 30, H. 2, 1906, p. 259.
- KRONTHAL. Die Neutralzellen des centralen Nervensystems. *Arch. f. Psychiat.*, Bd. 41, H. 1, 1906, p. 233.
- DELAMARE et TANASESCO. Étude sur les artères du sympathique céphalique, cervical, thoracique et abdominal. *Journ. de l'Anat. et de la Physiol.*, mars-avril 1906, p. 97.
- ALEXANDER BRUCE. Distribution of the Cells in the Intermedio-lateral Tract of the Spinal Cord. *Trans. Royal Soc. of Edin.*, Vol. xlv., Part 1, 1906, p. 105.
- RICCARDO FUA. Il Neurone. *Annuario del Manicomio Provinciale di Ancona*, Ann. iii., Marchetti, Ancona, 1906.
- LA SALLE ARCHAMBAULT. Le faisceau longitudinal inférieur et le faisceau optique central. *Nouv. Icon. de la Salpêtrière*, jan.-fév. 1906, p. 103.
- RUDOLF MEYER. Untersuchungen über den feineren Bau des Nervensystems der Asteriden. *Zeit. f. Wissenschaft Zool.*, Bd. 81, H. 1, 1906, p. 96.
- BOUGHTEN. The Increase in the Number and Size of the Medullated Fibres in the Oculomotor Nerve of the White Rat and of the Cat at Different Ages. *Journ. Comp. Neurol. and Psychol.*, March 1906, p. 153.
- RAVENNA. Sulla colorabilità primaria del tessuto nervoso in rapporto allo stato di ibernazione e de veglia. *Riv. di Patol. nerv. e ment.*, Vol. xi., f. 1, 1906, p. 1.

## PHYSIOLOGY

- PHELPS. Function of the left prefrontal lobe. *Am. Journ. of Med. Sci.*, March 1906, p. 457.
- SHERKINGTON. Observations on the Scratch-reflex in the Spinal Dog. *Journ. Physiol.*, March 13, 1906, p. 1.
- F. H. SCOTT. On the Relation of Nerve Cells to Fatigue of their Nerve Fibres. *Journ. Physiol.*, March 13, 1906, p. 145.
- TEBB. The Cholesterin of the Brain. *Journ. Physiol.*, March 13, 1906, p. 106.
- ROSENHEIM. On the Preparation of Cholesterin from Brain. *Journ. Physiol.*, March 13, 1906, p. 104.
- KEITH LUCAS. On the Conducted Disturbance in Muscle. *Journ. Physiol.*, March 13, 1906, p. 51.
- PRÉVOST et MIONI. L'anémie cérébrale modifiant la crise épileptiforme provoquée par le courant alternatif. *Ann. d'Électrobiol.*, No. 2, 1906, p. 81.
- FRUGONI e PEA. Intorno al centro e ai nervi secretori del rene. *Sperimentale*, Anno lx., f. 1, 1906, p. 136.
- MARGARET WASHBURN and MADISON BENTLEY. The Establishment of an Association Involving Colour-Discrimination in the Creek Chub, *Semotilus atromaculatus*. *Journ. Comp. Neurol. and Psychol.*, March 1906, p. 113.

## PSYCHOLOGY

- GEISSLER. Persönlichkeitsgefühl, Empfindung, Sein und Bewusstsein. *Arch. f. gesamte Psychol.*, March 1906, p. 33.
- LIPPS. Über "Urteilsgefühle." *Arch. f. gesamte Psychol.*, March 1906, p. 1.

- GRASSET. Le Psychisme inférieur. "Étude de physiopathologie clinique des centres psychiques," Chevalier et Rivière, Paris, 1906, 9 fr.  
 BARDOUX. Essai d'une psychologie de l'Angleterre contemporaine. F. Alcan, Paris, 1906, fr. 7.50.

## PATHOLOGY

- MÜNZER und FISCHER. Gibt es eine autogene Regeneration der Nervenfasern? *Neurol. Centralbl.*, März 16, 1906, S. 253.  
 RAIMANN. Zur Frage der autogenen Regeneration der Nervenfasern. *Neurol. Centralbl.*, März 16, 1906, S. 263.  
 JON G. LACHE. Altérations cadavériques des neurofibrilles. *Rev. Neurol.*, mars 15, 1906, p. 209.  
 HILL and MOTT. The neuro-fibrils of the large ganglion cells of the motor cortex of animals in which the four arteries had been ligatured to produce cerebral anæmia. (*Proc. Physiol. Soc.*) *Journ. Physiol.*, March 13, 1906, p. iv.  
 JULES DONATH. Preuve de l'existence de la choline dans le liquide céphalo-rachidien à l'aide du microscope polarisant. *Rev. Neurol.*, fév. 28, 1906, p. 145.  
 BALOGH. Beiträge zur Bestimmung des cytodagnostischen Wertes des Liquor cerebrospinalis. *Wien. med. Woch.*, Nr. 9, 1906, p. 418.  
 SPIELMEYER. Ueber das Verhalten der Neuroglie bei tabischen Optikus-atrophie. *Klin. Monatsblätter f. Augenheilk.*, Feb.-März 1906, p. 97.  
 FORD ROBERTSON. The Pathology of General Paralysis of the Insane. *Rev. Neurol. and Psychiat.*, March 1906, p. 169.  
 JOHN TURNER. The Pathology of Epilepsy. *Brit. Med. Journ.*, March 3, 1906, p. 496.  
 LEJONNE. Lésions des Cellules des Cornes Antérieures dans la Sclérose en Plaques à forme amyotrophique. (*Soc. de Neurol.*) *Rev. Neurol.*, fév. 28, 1906, p. 179.  
 HERZOG. Über die Sehbahn, das Ganglion opticum basale und die Fasersysteme am Boden des dritten Hirnventrikels in einem Falle von Bulbusatrophie beider Augen. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 30, H. 3-4, 1906, S. 223.  
 KÖLPIN. Erweichungsherde in der Medulla oblongata mit retrogenen Degenerationen in Pyramidenbahn und Schleife. *Arch. f. Psychiat.*, Bd. 41, H. 1, 1906, p. 286.  
 STRÄUSSLER. Über eigenartige Veränderungen der Ganglienzellen und ihrer Fortsätze in Centralnervensystem eines Falles von kongenitaler Kleinhirnatrophie. *Neurol. Centralbl.*, März 1, 1906, S. 194.  
 DERCUM. Thyroid Metastasis to the Spine. *Journ. Nerv. and Ment. Dis.*, March 1906, p. 153.

## CLINICAL NEUROLOGY AND PSYCHIATRY

## GENERAL—

- SAVAGE. An Address on the Borderland of Insanity. *Brit. Med. Journ.*, March 3, 1906, p. 489.  
 SCHULTZ. Gehirn und Seele. J. A. Barth, Leipzig, 1906, M. 5.  
 MOTT and HALLIBURTON. The Suprarenal Capsules in Cases of Nervous and other Diseases. (*Proc. Physiol. Soc.*) *Journ. Physiol.*, March 13, 1906, p. iii.  
 ANTON. Über den Wiedersatz der Funktion bei Erkrankungen der Gehirns. S. Karger, Berlin, 1906, M. 0.75.

## MUSCLES—

- ARMAND-DELILLE et ALBERT-WEIL. Syndrome Myopathique chez un enfant de 7 ans. (*Soc. de Neurol.*) *Rev. Neurol.*, fév. 28, 1906, p. 190.  
 C. MACFIE CAMPBELL. A Case of Muscular Dystrophy affecting Hands and Feet; Depression after Exhaustion, with Recovery. *Rev. Neurol. and Psychiat.*, March 1906, p. 192.

## PERIPHERAL NERVES—

- JAMES SHERRAN. The Erasmus Wilson Lectures on the Distribution and Recovery of Peripheral Nerves Studied from Instances of Division in Man. *Lancet*, March 17, 1906, p. 727.  
 LAFAGE. Contribution à l'étude des paralysies névritiques de la coqueluche. Dirion, Toulouse, 1906, 2 fr.

- KUTTNER. Ueber corticale Herderscheinungen in der amnestischen Phase polyneuritischer Psychosen. *Arch. f. Psychiat.*, Bd. 41, H. 1, 1906, p. 134.
- INGELRANS. Névralgies et névrites diabétiques. *Gaz. des Hôp.*, mars 3, p. 303.
- BEDUSCHI. Le forme fruste della neurite interstiziale ipertrofica e progressiva dell' infanzia. *Riv. di Patol. Nerv. de Ment.*, Vol. xi., f.i., 1906, p. 10.
- DUMAREST. Des névroses et névrites du pneumogastrique chez les tuberculeux. Gainche, Paris, 1906.
- NINA-RODRIGUES. La psychose polynévritique et le bérubéri. *Ann. méd.-psychol.*, mars-avril 1906, p. 177.
- ARMAND-DELILLE et DENECHÉAU. Syndrome de Landry avec lymphocytose du liquide céphalorachidien. (Soc. de Neurol.) *Rev. Neurol.*, fév. 28, 1906, p. 191.

## SPINAL CORD—

- SEIFFER. Spinales Sensibilitätschema für die Segmentdiagnose der Rückenmarkskrankheiten zum Einzeichnen der Befunde am Krankenbett. Hirschwald, Berlin, 1906, M. 1.20.
- KRAUSS. A Case of Brown-Sequard Paralysis, due to a Fall upon the Head; Operation; Autopsy. *Journ. Nerv. and Ment. Dis.*, March 1906, p. 173.
- GRINKE. Three Cases of Traumatic Brown-Séquard Paralysis. *Am. Journ. of Med. Sci.*, March 1906, p. 486.
- ROBBINS. A Peculiar Case of Infantile Palsy of Spinal Origin. *New York Med. Journ.*, March 10, p. 506.
- Progressive Muscular Atrophy.**—CHARLES L. DANA. Progressive Muscular Atrophy; a Study of the Causes and Classification. *Journ. Nerv. and Ment. Dis.*, Feb. 1906, p. 81.
- Poliomyelitis Anterior Acuta.**—ELLERMANN. Über den Befund von Rhizopoden bei zwei Fällen von Poliomyelitis acuta. *Centralbl. f. Bakteriöl., Parasiten und Infektionskrankheiten*, März 10, 1906, p. 648.
- Tabes.**—HÜBNER. Zur Tabes-Paralyse-Syphilis-Frage. *Neurol. Centralbl.*, März 16, 1906, S. 242.
- LAPINSKY. Einige wenig beschriebene Formen der Tabes dorsalis. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 30, H. 3-4, 1906, S. 178.
- MALAISE. Die Prognose der Tabes dorsalis. S. Karger, Berlin, 1906, M. 1.
- Friedreich's Ataxia.**—BALLET et TAGUET. Maladie familiale. Maladie de Friedreich ou Hérédo-ataxie cérébelleuse. (Soc. de Neurol.) *Rev. Neurol.*, fév. 23, 1906, p. 207.
- CHIADINI. Un Caso di Malattia di Friedreich. *Rev. Crit. di Clin. Med.*, Marzo 10, p. 149.
- A. H. DODGE. An Isolated Case of Friedreich's Ataxia. *Journ. of Am. Med. Ass.*, March 17, p. 802.
- LECOUFFE. De l'origine hérédo-syphilitique de certains cas de maladie de Friedreich. (Thèse.) Le Bigot Frères, Lille, 1906.
- Syringomyelia.**—LEENHARDT et NORERO. Sur un cas de Syringomyélie à prédominance unilatérale avec Atrophie Musculaire à topographie radiculaire. (Soc. de Neurol.) *Rev. Neurol.*, fév. 28, 1906, p. 177.
- Pott's Disease.**—ALQUIER. Les principales formes des troubles nerveux dans le mal de Pott sans gibbosité. *Nouv. Icon. de la Salpêtrière*, jan.-fév. 1906, p. 2.
- Combined Sclerosis.**—VERGER et GRENIER DE CARDENAL. Un cas de sclérose combinée pseudo-systématique. *Rev. Neurol.*, mars 15, 1906, p. 212.
- Conus.**—LOEB. Gutachten über eine traumatische Verletzung des Conus terminalis. *Mitteil. aus der Grenzgebieten der Med. und Chir.*, Bd. 15, H. 5, p. 413.
- Spine.**—PIERRE MARIE et LÉRI. La spondylose rhizomélique; anatomie pathologique et pathogénie. *Nouv. Icon. de la Salpêtrière*, jan.-fév. 1906, p. 82.
- BRUINE. Chronische Steifigkeit der Wirbelsäule. Breitkopf & Härtel, Leipzig, 1906, M. —75.

## BRAIN—

- W. G. SPILLER. Separate Sensory Centres in the Parietal Lobe for the Limbs. *Journ. Nerv. and Ment. Dis.*, Feb. 1906, p. 117.
- Meningitis.**—FERRARD. Le diagnostic de la meningite cérébro-spinale épidémique. *Gaz. des Hôp.*, mars 10, p. 339, et mars 17, p. 375.
- OSTERMANN. Die Meningococcenpharyngitis als Grundlage der epidemischen Genickstarre. *Deutsch. med. Woch.*, März 15, p. 414.
- Hydrocephalus.**—L. W. WEBER. Zur Symptomatologie und Pathogenese des erworbenen Hydrocephalus internus. *Arch. f. Psychiat.*, Bd. 41, H. 1, 1906, p. 64.

- Vascular Lesions.**—RUSSELL. Cerebral Manifestations of Hypertonus in Sclerosed Arteries. *Practitioner*, March 1906, p. 806.  
 J. CAMERON TURNBULL. Intracranial Hemorrhage in the Newborn. *Brit. Med. Journ.*, March 24, 1906, p. 677.  
 E. FARQUHAR BUZZARD and JOSEPH CUMMING. A Case of Post-traumatic Hemorrhage from the Superior Longitudinal Sinus without Fracture of the Skull; Operation and Recovery. *Lancet*, March 24, 1906, p. 822.  
 STARR. Intracranial Lesion as sequelæ of Chronic Purulent Otitis media. *Med. Rec.*, March 10, 1906, p. 369.  
 STODDART BARR. Notes of a Fatal Case of Septic Thrombosis of the Lateral Sinus, Secondary to Chronic Otitis Media Purulenta in the Left Ear. *Lancet*, March 24, 1906, p. 823.  
 PERAZZOLO. Su un caso di aneurisma dell'arteria comunicante posteriore. *Riv. di Patol. nerv. e ment.*, Vol. xi, f. 1, 1906, p. 17.  
 MOURET. A propos d'un cas de complications endocraniennes consécutives à une otite moyenne suppurée. (Thèse.) Firmin, Montane et Sicardi, Montpellier, 1905.  
 COMBY. L'Encéphalite aiguë chez les enfants. Gainche, Paris, 1906.  
**Hemiplegia.**—FARQUHAR BUZZARD and STANLEY BARNES. A Case of Chronic Progressive Double Hemiplegia. *Rev. Neurol. and Psychiat.*, March 1906, p. 182.  
**Tumour.**—C. FÜRSTNER. Ueber die operative Behandlung der Gehirngeschwulste. *Arch. f. Psychiat.*, Bd. 41, H. 1, 1906, p. 202.  
 J. LINDSAY STEVEN. Case of Round-Cell Sarcoma of the Brain situated in the Frontal Lobes and beginning with Mental Symptoms. *Glasg. Med. Journ.*, March 1906, p. 170.  
 OSTERWALD. Beitrag zur Diagnose des Cysticercus ventriculi quarti. *Neurol. Centralbl.*, März 16, 1906, S. 265.  
 DERCUM. Sarcoma of the Cerebellum; Sarcomatous Infiltration of the Spinal Pia. *Journ. Nerv. and Ment. Dis.*, March 1906, p. 169.  
 RAYMOND et LEJONNE. Syndrome de Compression Cérébrale et radiculoganglionnaire par hypertension du liquide céphalorachidien dans un cas de Tumeur du Cervelet. (Soc. de Neurol.) *Rev. Neurol.*, fév. 28, 1906, p. 198.  
 LARUELLE. Sarcome du lobe droit du Cervelet et du Pédoncule Cérébelleux inférieur droit. Valeur diagnostique de la position de la tête. (Soc. de Neurol.) *Rev. Neurol.*, fév. 28, 1906, p. 204.  
**Gliosis.**—BULLARD and SOUTHARD. Diffuse Gliosis of the Cerebral White Matter in a Child. *Journ. Nerv. and Ment. Dis.*, March 1906, p. 188.  
**Syphilis.**—STRÄUSSLER. Zur Lehre von der miliaren disseminierten Form der Hirnlues und ihrer Kombination mit der progressiven Paralyse. *Monatssch. f. Psychiat. u. Neurol.*, März 1906, p. 244.  
**General Paralysis.**—MONGERI. Contribution à l'étude de l'étiologie de la paralysie progressive. *Centralbl. f. Nervenheilk. u. Psychiat.*, März 1, 1906, S. 169.  
 NÄCKE. Erbllichkeit und Prädisposition nach Degenerationen bei der progressiven Paralyse der Irren. *Arch. f. Psychiat.*, Bd. 41, H. 1, 1906, p. 295.  
**Amaurotic Family Idiocy.**—QUACKENBOSS. Amaurotic Family Idiocy. *Boston Med. and Surg. Journ.*, March 1, 1906, p. 238.  
 RIVA. Idiozia cerebroplegica familiare e microcefalia. *Annuario del Manicomio Provinciale di Ancona*, Ann. iii, Marchetti, Ancona, 1905.  
**Bullet Wound.**—LAWFORD KNAGGS. A Clinical Lecture on Two Cases of Bullet Wound of the Brain. *Lancet*, March 3, 1906, p. 581.

#### MENTAL DISEASES—

- EDWARD COWLES. The Problem of Psychiatry in the Functional Psychoses. *Am. Journ. of Insanity*, Oct. 1905, p. 189.  
 SOUTZO (fil.). La psychiatrie moderne et l'œuvre du professeur Kraepelin. *Ann. méd.-psychol.*, mars-avril 1906, p. 243.  
 PILCZ. Beitrag zur vergleichenden Rassen-Psychiatrie. F. Deuticke, Vienna, 1906, M. 2.50.  
 FRANZ and HAMILTON. The Effects of Exercise upon the Retardation in Conditions of Depression. *Am. Journ. Insan.*, Oct. 1905, p. 239.  
 A. SCHOTT. Simulation und Geistesstörung. *Arch. f. Psychiat.*, Bd. 41, H. 1, 1906, p. 254.

- RAECKE. Zur Symptomatologie des epileptischen Irreseins, in besonders über die Beziehungen zwischen Aphasie und Perseveration. *Arch. f. Psychiat.*, Bd. 41, H. 1, 1906, p. 1.
- WOLLENBERG. Die Melancholie. *Deutsche Klinik*, H. 158, 1906, p. 493.
- CLARENCE FARRER. Dementia Præcox in France, with some References to the Frequency of this Diagnosis in America. *Am. Journ. Insan.*, Oct. 1906, p. 257.
- THEODORE A. HOCH. A Study of Somatic Ideas in Various Psychoses. *Am. Journ. Insan.*, 1906, p. 283.
- GEORGES BLIN. Les troubles oculaires dans la démence précoce. *Rev. Neurol.*, fév. 28, 1906, p. 151.
- DECROLY. Contribution au diagnostic des irrégularités. Les frontières anthropométriques des anormaux d'après M. Binet. *Journ. de Neurol.*, fév. 20, 1906, p. 61.
- MORAVCSIK. Künstlich hervorgerufene Halluzinationen. *Centralbl. f. Nervenheilk. u. Psychiat.*, März 15, 1906, S. 209.
- JULIUSBÜRGER. Ueber Pseudo-Melancholie. *Centralbl. f. Nervenheilk. u. Psychiat.*, März 15, 1906, S. 216.
- RIVA. Il vitto degli alienati. La somministrazione del vino agli alienati. *Annuario del Manicomio Provinciale di Ancona*, Anno iii., Marchetti, Ancona, 1906.
- BLEULER. Affektivität, Suggestibilität, Paranoia. Carl Marhold, Halle a. S., 1906, M. 3.
- BESSIERE. Les stéréotypies démentielles. *Ann. méd.-psychol.*, mars-avril 1906, p. 206.
- GIMBAL. Les incendiaires (suite). *Ann. méd.-psychol.*, mars-avril 1906, p. 214.
- LUCIEN LAGRIFFE. Considérations sur quelques degrés de la responsabilité. *Ann. méd.-psychol.*, mars-avril 1906, p. 229.
- MARGUERY. Des effets de l'insanité de l'esprit sur la capacité civile. Rousseau, Paris, 1906, 4 fr.
- ALLONNES et JUQUELIER. Délire de persécution à trois, avec séquestration volontaire. F. Alcan, Paris, 1906.
- TOURENG. État mental des Incendiaires. Michalon, Paris, 1906, fr. 2.50.
- TREPSAT. Œdème des pieds chez deux imbéciles. *Nouv. Icon. de la Salpêtrière*, jan.-fév. 1906, p. 95.
- Alcohol.**—PFAFF. Die Alkoholfrage vom ärztlichen Standpunkt. Reinhardt, München, 1906, M. 1.
- CHARRA. Contribution à l'étude de l'alcoolisme héréditaire. Rey, Lyons, 1906, 2 fr.
- BONHOEFFER. Die alkoholischen Geistesstörungen. *Deutsche Klinik*, H. 158, 1906, p. 510.
- E. L. HUNT. Korsakoff's Disease. *Med. Rec.*, March 10, 1906, p. 387.

#### GENERAL AND FUNCTIONAL DISEASES—

- Chorea.**—GUSTAVO MODENA. Su di un caso di corea di Huntington. *Annuario del Manicomio Provinciale di Ancona*, Anno iii., Marchetti, Ancona, 1906.
- Epilepsy.**—SPRATLING. Unrecognised Epilepsy. *Journ. of Amer. Med. Ass.*, March 10, 1906, p. 722.
- PLAVEC. Kleine motorische Epilepsie. *Neurol. Centralbl.*, März 1, 1906, S. 207.
- Hysteria.**—HERM. H. HOPPE. Hysterical Stigmata Caused by Organic Brain Lesions. *Journ. Nerv. and Ment. Dis.*, Feb. 1906, p. 101.
- VON VOSS. Zur Lehre vom hysterischen Fieber. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 30, H. 3-4, 1906, S. 166.
- STRÜMPPELL. Über das sogenannte hysterische Fieber. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 30, H. 3-4, 1906, S. 281.
- DEROUBAIX. Le rire et le pleurer spasmodiques. *Journ. de Neurol.*, mars 5, 1906, p. 81.
- Neurasthenia.**—P. C. SMITH. Neurasthenia, Degeneracy, and Mobile Organs. *Brit. Med. Journ.*, March 3, 1906, p. 494.
- GUTHRIE RANKIN. The Treatment of Neurasthenia. *Brit. Med. Journ.*, March 3, 1906, p. 492.
- Exophthalmic Goitre.**—STRANSKY. Zur Antithyreoidinbehandlung der Basedow'schen Krankheit. *Wien. med. Press*, März 11, 1906, p. 510.

**Torticollis.**—STEYERTHAL. Zur Geschichte des Torticollis spasmodicus. *Arch. f. Psychiat.*, Bd. 41, H. 1., 1906, p. 29.

#### SPECIAL SENSES AND CRANIAL NERVES—

TALMAY. A Contribution to the Study of Pseudoneuritis optica. *New York Med. Journ.*, March 3, 1906, p. 442.

DREYFUS. Ueber traumatische Pupillenstarre. *Münch. med. Woch.*, März 27, p. 604.

WALTER H. HAW. An Attempt to Simplify the Diagnosis of Ocular Paralysis. *Lancet*, Feb. 24, 1906, p. 514.

#### MISCELLANEOUS SYMPTOMS—

SANDBERG. Über die Sensibilitätsstörungen bei cerebralen Hemiplegien. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 30, H. 3-4, 1906, S. 149.

C. K. MILLS. A Case of Crural Monoplegia, representing the early stage of a Unilateral Ascending Paralysis due to the Degeneration of the Pyramidal Tracts. *Journ. Nerv. and Ment. Dis.*, Feb. 1906, p. 115.

ACHARD et RIBOT. Rhumatisme déformant du côté opposé à l'Hémiplégie. (Soc. de Neurol.) *Rev. Neurol.*, fév. 28, 1906, p. 194.

ERB. Ein weiterer Fall von angiosklerotischer Bewegungsstörung des Arms. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 30, H. 3-4, 1906, S. 201.

NEWMARK. A Case of Ascending Unilateral Paralysis. *Journ. Nerv. and Ment. Dis.*, March 1906, p. 182.

EGGER et RAYMOND. L'Acroparesthésie. Une lésion du Cordon Postérieur. (Soc. de Neurol.) *Rev. Neurol.*, fév. 28, 1906, p. 174.

GRAEFFNER. Einige Studien über Reflexe, besonders an Hemiplegikern. *Münch. med. Woch.*, März 18, p. 489.

BAUMANN. Ueber den Rachenreflex. *Münch. med. Woch.*, März 27, p. 593.

LAPINSKY. Über die Herabsetzung der reflektorischen Vorgänge im gelähmten Körperteil bei Kompression der oberen Teile des Rückenmarks. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 30, H. 3-4, 1906, S. 239.

DEJERINE et NORERO. Épilepsie Spinale vraie et Clonus de la rotule chez une Hystérique anorexique ayant été atteinte d'une Hémiplégie gauche actuellement guérie. (Soc. de Neurol.) *Rev. Neurol.*, fév. 28, 1906, p. 182.

KEMPNER. Ueber Störungen im Augengebiet des Trigeminus spezielle des Cornealreflexes und ihre diagnostische Verwerfung. *Berlin klin. Woch.*, März 26, p. 379.

ACHARD et RIBOT. Troubles de la Motilité des membres inférieurs rappelant ceux de l'Ataxie Cérébelleuse. (Soc. de Neurol.) *Rev. Neurol.*, fév. 28, 1906, p. 193.

CASTEX. Le réflexe patellaire est indépendant de la surface du percuteur. *Journ. de Neurol.*, mars 5, 1906, p. 89.

DARCANNE. Le signe de Kernig dans la paralysie générale. *Journ. de Neurol.*, mars 5, 1906, p. 91.

BELBEZE. Sur la présence du signe de Kernig dans la zona. *Arch. gén. de méd.*, fév. 27, 1906, p. 520.

RUGGLES. Observations on Ganser's Symptom. *Am. Journ. Insan.*, Oct. 1905, p. 307.

THORP. Du vertige voltaïque; Son application au diagnostic des fractures anciennes du rocher. Gout et Cie, Orléans, 1906.

DOUGLAS BRYAN. Recurrent Herpes Gestationis. *Lancet*, Feb. 24, 1906, p. 512.

BRISAUD et MOUTIER. Cyphose prononcée chez un tuberculeux. *Nouv. Icon. de la Salpêtrière*, jan.-fév. 1906, p. 30.

SÉRIEUX et MIGNOT. Observation clinique d'un cas d'amnésie rétro-antérograde consécutive à la pendaison. F. Alcan, Paris, 1905.

LIEPMANN. Der weitere Krankheitsverlauf bei dem einseitig Apraktischen und der Gehirnbefund auf Grund von Serienschnitten. S. Karger, Berlin, 1906, M. 1.50.

MARCEL NATHAN. Note sur un cas d'Amusie incomplète chez un musicien professionnel atteint également d'aphasie sensorielle très atténuée. (Soc. de Neurol.) *Rev. Neurol.*, fév. 28, 1906, p. 202.

HENRI LAMY. Troubles d'Élocution chez un ancien Aphasique. (Soc. de Neurol.) *Rev. Neurol.*, fév. 28, 1906, p. 186.

ALFRED GORDON. On Retrograde Amnesia. *New York Med. Journ.*, March 3, 1906, p. 440.



- POCHHAMMER. Beobachtungen über Entstehung und Rückbildung traumatischer Aphasie. *Mitteil. aus den Grenzgebieten der Med. und Chir.*, Bd. 15, H. 5, 1906, p. 495.
- OSKAR FISCHER. Über Makropsie und deren Beziehungen zur Mikrographie, sowie über eine eigentümliche Störung der Lichtempfindung. *Monatssch. f. Psychiat. u. Neurol.*, März 1906, p. 290.
- KLEIST. Über Apraxie. *Monatssch. f. Psychiat. u. Neurol.*, März 1906, p. 289.
- LIEPMANN. Der weitere Krankheitsverlauf bei dem einseitig Apraktischen und der Gehirnbefund auf Grund von Serienschnitten. *Monatssch. f. Psychiat. u. Neurol.*, März 1906, p. 217.

#### TREATMENT\*—

- OPPENHEIM. Psychotherapeutische Briefe. S. Karger, Berlin, 1906, M. 1.
- NEUMANN. Die Heilung der Nervosität durch intelligente Lebenszucht und rationelle Lebenshaltung. Borggold, Leipzig, 1906, M. 1.20.
- HIRSCHL. Bemerkungen zur Behandlung des Morbus Basedowii. *Wien. klin. Woch.*, März 15, 1906, p. 295.
- WHITCOMBE BROWN. The Therapeutic Effects of Mercury in a case of Tuberculous Meningitis. *Lancet*, March 24, 1906, p. 825.
- ABADIE et DUPUY-DUTEMPS. Hémispasme Faciale guéri par une Injection profonde d'Alcool. (Soc. de Neurol.) *Rev. Neurol.*, fév. 28, 1906, p. 197.
- KALISCHER. Über das Schlafmittel Proponal. *Neurol. Centralbl.*, März 1, 1906, S. 206.
- BRUEL. Traitement des chorées et des tics de l'enfance. Steinheil, Paris, 1906.
- J. R. EASTMANN. The Effects of Osmic Acid Injections. *Journ. of Am. Med. Ass.*, Feb. 24, p. 556.
- TUBBY. The Hunterian Oration on Recent Surgical Methods in the Treatment of Certain Forms of Paralysis. *Brit. Med. Journ.*, March 3, 1906, p. 481.
- SPILLER, FRAZIER and KAATHOVEN. Treatment of selected cases of cerebral, spinal and peripheral nerve palsies and athetosis by Nerve Transplantation. *Am. Journ. of Med. Sci.*, March 1906, p. 430.
- JORDAN LLOYD. On Facial Neuralgia and its Curative Treatment by Excision of the Gasserian Ganglion. *Birm. Med. Rev.*, Feb. 1906, p. 57.
- HOFMEISTER und MEYER. Operierter Tumor des Ganglion Gasseri. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 30, H. 3-4, 1906, S. 216.

\* A number of references to papers on Treatment are included in the Bibliography under the individual Diseases.

# **Review**

of

# **Neurology and Psychiatry**

---

## **Original Articles**

### **LESIONS OF THE LEFT FIRST TEMPORAL CONVOLUTION IN RELATION TO SENSORY APHASIA.**

By WILLIAM G. SPILLER, M.D.,

Professor of Neuropathology and Associate Professor of Neurology in the  
University of Pennsylvania.

From the Department of Neurology and the Laboratory of Neuropathology  
of the University of Pennsylvania, and from the  
Philadelphia General Hospital.

THE centre for word-hearing in right-handed persons is generally believed to be in the posterior part of the left first temporal convolution and possibly of the second. According to Bastian, the merit of having determined this region belongs to Wernicke, but Bastian thinks that disturbance (Bastian, "Aphasia and Other Speech Defects." H. K. Lewis, London, 1898) to a very marked extent of the functions of the visual word centre as a result of a lesion in the upper temporal convolutions is not so universal as Wernicke and Dejerine believed. Thus out of sixteen recorded cases of sensory aphasia in which the lesion was pretty closely limited to the hinder part of the first, and more or less of the second temporal convolutions, in only five is any mention of some amount of word-blindness. In the sixteen cases to which Bastian referred, motor aphasia existed in six, some paraphasia in six, aphasia and paraphasia in one, and voluntary speech seemed to be, but to a less extent, affected in three.

Von Monakow says that so many instances of word-deafness, resulting from a lesion of the left first temporal convolution in right-handed persons are recorded, that the relation between this lesion and the word-deafness cannot be doubted. There are cases of lesion confined to the left second and third temporal convolutions with sensory aphasia, and others with a lesion confined to the posterior part of the left first temporal convolution without aphasia, and in illustration of the latter statement he refers to Byrom Bramwell's Case 10, *Brain*, 1899 (Von Monakow, "Gehirnpathologie," 2nd edition, p. 929).

Still, word-deafness with destruction of the posterior part of the left first and second temporal convolutions is not so common that new cases may be ignored, and the two following cases are reported to form a contrast with the third, in which destruction of the left first temporal convolution in a right-handed man did not cause word-deafness.

CASE I. William Jones, aged 29 years, was admitted to the Polyclinic Hospital, to the service of Dr Steinbach, May 12, 1901, with the diagnosis of fractured skull. He was struck in the left temporal region with a base-ball bat. When he was admitted he had profuse hæmorrhage from the left ear, and this continued most of the night. He has been unconscious since admission, moves his limbs somewhat, and has tried to get out of bed, but does not seem to know when anyone is speaking to him. He is on liquid diet, and only a small amount can be forced into him. The bowels have been opened by enema. The temperature is  $100\frac{1}{4}^{\circ}$ ; pulse, 60; respiration, 14. Hæmorrhage from the ear has entirely ceased, and he lies in a semi-comatose state most of the time.

May 17.—Paralysis, both motor and sensory, has appeared in the right limbs. The pulse is slow, and more feeble than it was previously. He is still unconscious, but less restless than before.

May 18.—He seems to understand some things that are said to him, but he cannot do more than occasionally mutter a reply. There is slight motor power in the right leg. The left eye is inflamed, the right iris responds to light.

May 20.—Paralysis in the right leg is disappearing. He tries to answer questions, but cannot make himself understood.

May 22.—He sings occasionally, but cannot make himself understood; seems to understand some things said to him.

May 24.—His right arm moved to-day; he has twitchings of the right angle of the mouth, otherwise his condition is about the same.

May 26.—He has regained some use of his right arm.

June 3.—A slight twitching of the mouth is noticed on the right side. He speaks when spoken to, but replies by meaningless words.

July 6.—*Notes by Dr Spiller.*—The tic of the right side of face in its lower portion still continues, and probably is caused by a large scar on the right side of the neck, extending from the middle of the clavicle to the mastoid process. The paralysis on the right side of the body has almost entirely disappeared. His speech is as follows: "Well, sir, I know I use to have good. I can ever do ever know. Yes I have been plenty; I have been impose; I know where I at, who I not; I would to work to work plenty plenty." This is mingled with words that cannot be understood. All this was in answer to the question of his name. He does not recognise his name when it is mentioned among other names. When told to sit up in bed he did so; when told to lie down, he did so; when told to give his hand he did not seem to understand; he sat up a second time on command, when all gestures were avoided. He was probably word-blind.

June 15.—An operation over the posterior part of the left first temporal convolution was advised. Dr Steinbach made a horse-shoe shaped incision (base downward) over the temporal region, dissected the periosteum from the bone, exposing a stellate fracture; and at the superior border of the squamous portion of the temporal bone a depression was seen. The patient was trephined just above the point of depression, and the opening was enlarged by rongeur forceps to about the size of half a dollar. The dura was found torn and the brain substance protruding. The finger, when inserted, entered a large cavity filled with degenerated brain material and blood-clots.

After removal of this broken-down tissue, the cavity was irrigated with normal salt solution, and a silk thread was inserted as a drain. At this stage hæmorrhage started, and the cavity was packed with iodoform gauze. The scalp wound was now brought together and sutured.

June 16.—The patient had a very comfortable day. He does not show any signs of improvement as yet.

June 17.—He still remains word-deaf. No weakness of the right side is detected. General condition is good.

June 24.—The wound was re-dressed to-day, and all the packing was removed. The wound is in an excellent condition. The patient is allowed to be up.

July 1.—The wound of the scalp has healed nicely, but there is no improvement in the mental condition of the patient. He was sent to the Philadelphia General Hospital, July 5th, 1901, and came into the service of Dr Spiller.

His word-deafness persisted unchanged. He occasionally understood a word here and there in a sentence, and guessed at the meaning of the rest of the sentence. After he had been in the nervous wards about a year, he became insane and was referred to the insane department.

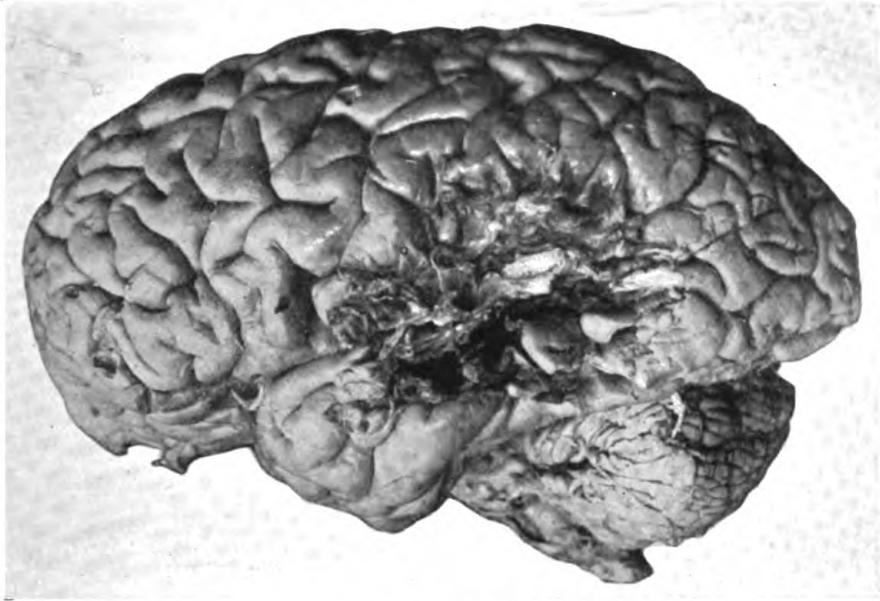
July 17, 1902.—He says he hears people talking to him, hears the devil and thinks he saw him also, that he jumps at him and wants to kill him.

July 19, 1902.—He no longer has auditory hallucinations, but has been unmanageable at times, and very abusive and violent, has refused to remain in bed, and has wandered about the grounds, and threatened the nurse several times.

August 25, 1902.—He has delusions of persecution, and has again shown evidence of hallucinations of hearing, which, with the visual hallucinations, are especially interesting on account of the injury of the centre for word-hearing and of the optic radiations.

I am indebted to Dr Charles W. Burr for the brain of this patient. An area of sclerosis implicates the posterior part of the first and second left temporal convolutions and lower part of the parietal lobe. It extends to the posterior part of the island of Reil, and to the optic radiations, and may implicate the latter.

The history in the second case is very brief. The man was in the service of the late Dr Frederick A. Packard, at the Pennsylvania Hospital, and the brain was given to me by Dr Packard and Dr Simon Flexner in April 1901. The former wrote that the man had had Cheyne-Stokes breathing many days before his



Brain in Case I.



Brain in Case III.



death, and was so deaf that it was impossible to obtain any but the most meagre history from him, and the deafness had been long-standing. He had no friends, consequently his former history could not be obtained.

The greater part of the left first and second temporal convolutions are sclerotic and the cortex here is extremely atrophied.

The third case, in contrast to the other two, is very interesting. The patient was under the care of Dr Herman B. Allyn, and the clinical notes are from him. I am much indebted to Dr Allyn for them.

October 14, 1900.—F. R. S., aged 57 years, complains of painful cramps in his right upper and lower limbs. The leg is jerked up in flexion and subsequent extension is slow and painful. His clinical history is as follows:—

On January 28, 1881, he fell in New York in an apoplectic form attack. He recovered with disturbance of speech and some loss of power on the right side of the body. Four years ago he had another seizure. He is now partly aphasic, cannot write without making mistakes, walks with difficulty, and at times has difficulty in swallowing and in urination; also has hiccough and vomiting spells.

November 29, 1902.—His condition is about the same as at the previous examination. His chief complaint is spasmodic twitching of the leg muscles and weakness of the ankles. He has diplopia, which is not constant, and difficulty in stepping down from the sidewalk to the street.

October 21, 1903.—He cannot walk without help. Speech is no worse. He has been obliged for years to open the anus with the finger in order to start a stool.

January 7, 1904.—He is weaker and cannot stand, as his legs bend under him and he does not know where they are when he is in bed. He has involuntary micturition, and painful spasms of the right leg.

In reply to questions, Dr Allyn informed me that the patient was never musical, had always had remarkably acute hearing, and there were no words he had difficulty in understanding. He did not swing or drag his right lower limb so long as Dr Allyn had had him under his observation—about four years. He had



some loss of power on the right side, and the right ankle was likely to give way under him and he was liable to fall. He always shook hands with his right hand. His gait was somewhat ataxic. He understood *all* that was said to him, but was frequently at a loss for a word in speaking, although he could carry on a conversation, tell anecdotes, and was very entertaining. When he was fatigued or was not feeling well his speech became at times muffled and indistinct, so that he would have to

July 15 1902

My dear Doctor

Please send me some more

tablets for treating of my ankles. as I am about out and the weather still goes on. My trip cost did me good and I start out well here, for the rest of the summer. We will not get into our new home until late in October. So we must go somewhere else after leaving here but better address to my son's office.

Letter written by the patient (Case III.).

be asked to repeat. When he had difficulty in saying a word, he would say, with an air of embarrassment and a slight laugh, "You know what I mean." His speech was probably much worse following his first attack in 1881 than at any time since. He could write legibly, but rarely did write, and was liable to misuse words in writing, and therefore had his letters supervised by a member of the family. In the letter reproduced there are several mistakes: "address" for "addressed," "tabled" for "tablets," "troubled" for "trouble." He was sound mentally, had good judgment and reasoning powers, but his memory had failed somewhat. He read constantly. He was right-handed.

The microscopical examination shows that the patient had tabes. The first left temporal convolution is entirely destroyed and appears as a shrivelled mass of tissue, and the sclerosis extends to the posterior limb of the internal capsule and to the posterior horn of the lateral ventricle, and implicates the optic radiations. The second left temporal convolution is intact, and the first temporal convolution on the right side is unusually well developed.

The only explanations that are suggested to me for the preservation of word-hearing in this case are that the centre for this function was largely in the posterior part of the left second temporal convolution, or that the right first temporal convolution had been unusually well developed during the patient's entire life, and was capable of assuming the function of the destroyed left first temporal convolution.

Some cases in literature give evidence that the right cerebral hemisphere may sometimes replace the left in the functions of speech, even in adults.

Freund reports the following case :—

A woman, right-handed, 73 years of age, had apoplexy, and following this, right hemiplegia and aphasia. Improvement occurred, and most important was the condition of word-hearing. She could distinguish between sounds, she understood what was read to her from the newspaper, she repeated sentences if they were short and spoken slowly to her. She did not have alexia, letters were written correctly, and she copied correctly. In spontaneous writing, and in writing to dictation, or after long copying, repetitions were frequent. The understanding of spoken words seemed to be impaired only when she was spoken to rapidly or in too long sentences, or in unusual words. Right lateral homonymous hemianopsia and the inability to name objects seen or touched by her indicated the location of the lesion.

At the necropsy an area of softening was found in the left temporal lobe, and had caused much destruction. The lesion extended to the middle of the occipital lobe posteriorly, and to about  $1\frac{1}{2}$  cm. behind the anterior end of the corpus callosum anteriorly.

Freund believed that the restoration of word-hearing in this case is to be explained by the assumption that the right temporal

lobe functionated in place of the destroyed left temporal lobe. Wernicke suggested a similar explanation for a case in 1874, and this view has been held by Pick, Kauders, V. Monakow, Entzian, and others. (Freund, *Neurologisches Centralblatt*, Oct. 1, 1904, p. 914, and Oct. 16, 1904, p. 965.)

In the discussion of this case, Sachs, of Breslau, expressed doubt concerning vicarious action of the right hemisphere in a person so well advanced in years, and Pick shared the doubt. Freund acknowledged that the objection was forcible, but because of the intense destruction of the left temporal lobe he assumed that the patient had since youth made partial use of her right temporal lobe in the function of word-hearing.

Bastian reports a case in which the whole of the left superior temporo-sphenoidal convolution was destroyed, with the exception of the anterior one-third,  $4\frac{1}{2}$  cm. in length. Of the middle temporo-sphenoidal convolution, the anterior 5 cm. were perfectly intact; but posteriorly, only a narrow portion of the inferior part of the convolution remained, and that was in a discoloured and degenerated condition. The left angular and marginal convolutions were destroyed. The patient understood what was said to him and could repeat all simple common words. His spontaneous speech was limited to his name and short affirmative or negative answers, or short phrases. He understood what he read. Bastian believed that the destruction had been gradual, and that a gradual development in the functional activities of the corresponding convolutions of the right hemisphere occurred. This case had been under observation eighteen years, and there had never been word-deafness or word-blindness. Presumably the man was right-handed. ("Aphasia and Other Speech Defects," by H. C. Bastian. H. K. Lewis, London, 1898.)

A case is reported by Jolly in which right hemiplegia and motor aphasia followed an apoplectic attack. Word-hearing for simple questions was preserved, but complex questions were not understood. Reading was impossible. The necropsy revealed destruction of Broca's convolution, and of the first and partially of the second left temporal convolutions. Word-hearing in great measure is said therefore to have been preserved, notwithstanding complete destruction of the left first temporal convolution, and Jolly thinks the right hemisphere functionated in place of the left, although the patient was 73 years old at the time of

the attack, and lived but a short time after the attack began. (Jolly, *Centralblatt für Nervenheilkunde und Psychiatrie*, Oct. 21, 1899, p. 593.)

The third case that I have reported can not be explained on the ground that the patient was left-handed, as Dr Allyn is sure that he was not. There is evidence that in left-handed persons the speech centres are in the right cerebral hemisphere. In a case reported by Joffroy, the patient was word-deaf, word-blind, paraphasic and agraphic, and an area of softening was found in the middle portion of the right first temporal convolution, with meningo-encephalitis about it. Only three cases of sensory aphasia, according to Joffroy (Kussmaul, Touche, Köster), with lesions in the right side of the brain have been reported, and Joffroy's case makes the fourth. The three cases occurred in left-handed persons. Joffroy's patient wrote and ate with the right hand, and those who had known him had not observed that he was left-handed. (Joffroy, *Revue Neurologique*, Jan. 31, 1902, p. 112.) Mills and Weisenburg have reported a case of word-blindness in a right-handed man, with the lesion in the right cerebral hemisphere. (Mills and Weisenburg, *Medicine*, Nov. 1905.)

The case of Bloch and Bielschowsky seems to show that a lesion of the anterior part of the left first and second temporal gyri may cause word-deafness, and that involvement of the posterior part of these gyri is not always present when word-deafness exists. In their patient the hearing of sounds was excellent, but that of words was completely lost. Reading and writing were not tested. A hæmorrhagic focus was found in the point of the left temporal lobe, and it extended backwards and downwards. (Bloch and Bielschowsky, *Neurologisches Centralblatt*, Aug. 15, 1898.)

We need more cases of word-deafness from lesions of the left first and second temporal convolutions in their anterior portion before we can locate the centre for word-hearing in this part of the brain.

Probst seems to have shown that the centre for music-hearing may be in the anterior part of the first left temporal gyrus. A patient of his had motor and sensory aphasia, she could repeat only a few words, and these conveyed no meaning, but she could sing and articulate the words in singing correctly. She recog-

nised songs with which she had been familiar, and sang them correctly after some one else, and was able to sing unknown tunes, but without the notes. (Probst, *Archiv für Psychiatrie*, vol. xxxii. No. 2, p. 387.) The anterior part of the first left temporal gyrus was not implicated in the lesion.

The explanation of the substitutional functioning of the right cerebral hemisphere applies to motor speech as well as sensory.

A case that Byrom Bramwell reports shows that acute and complete destruction of Broca's area, and of the anterior end of the left island of Reil, in a person who has always been right-handed, may produce merely a very temporary motor aphasia. Bramwell believed that in his case the cortical centre in the right hemisphere corresponding to Broca's convolution was more highly educated than it is in the great majority of right-handed persons, and was able to carry on immediately the functions of the left motor-vocal speech centre. According to his views, the right cerebral hemisphere has more to do with speech than is usually taught, and the speech centre and speech functions are bilaterally represented in the brain, but not to the same extent in each hemisphere.

A somewhat similar case has been reported by Collier, and probably other similar cases could be found in the literature. (Bramwell, *Brain*, Autumn 1898; Collier, *Lancet*, 1899.)

---

## **TWO CASES OF EMBRYOMA IN THE FRONTAL LOBE OF THE BRAIN.**

By R. G. ROWS, M.D.,

Pathologist to the County Asylum, Lancaster.

AMONGST the very rare tumours which may be found in the cranial cavity, are the dermoid cysts and the embryomata. These tumours are more frequently met with in the base of the skull and in the hypophysis; very seldom have they been found in the brain itself. Wilms (1), while searching through the literature on the subject, has discovered only a few cases which have been recorded by Weigert, Beck, Bonorden,

Gauderer, Rokitansky, Bruns, Virchow, and Ziegler. Beck has also described eight cases of embryomata which he found in the nasal and pharyngeal cavities.

These tumours all resembled each other in containing some embryonal structures, but their complexity varied very much in the different cases. The structures which were found included skin with hair and glands, fat, mucous tissues, ciliated mucous membrane, cartilage, bone, muscle, and nervous tissue. Beck's case contained some thyroid gland substance.

In the course of last year I met with two cases in which an embryoma was situated in the frontal lobe of the brain.

In the first case the tumour, which was about an inch in diameter, lay in the mesial portion of the first frontal convolution of the brain of an epileptic aged 77. This man had been in the asylum for fifteen years. He had suffered from frequent epileptic attacks, which left him irritable and sometimes dangerous, but there had been no symptoms which pointed to the presence of a tumour in the brain. This tumour was a very simple one, and contained only epithelium, fibrous tissue, fat, and cholesterine crystals.

The other tumour was about the size of a hen's egg, and was found in the left frontal lobe of an epileptic aged 73. He had been in the asylum for thirty-eight years. His attacks occurred about once a month; he was often troublesome for a day or two before the attack, and excited and confused for two or three days after, but here again there were no symptoms of cerebral tumour. Before admission he had been an overlooker in a mill, and while here he worked, at different times, at tailoring, shoemaking, and mattress-making. He became demented gradually.

This tumour lay above the pia mater of the orbital surface of the left frontal lobe. The pia here was much thickened and had a pearly-white appearance.

The centre of the tumour was occupied by a soft, yellowish-grey substance, which consisted of fat cells, shed epithelial cells, and crystalline plates. In its wall were found skin, hair, sebaceous glands, embryonal blood-vessels, cartilage, bone, elastic tissue, and muscle.

It will be seen that the embryonal structures which were present in these two tumours differed very greatly. In the smaller one there was little more than a layer of epidermis;

while in the other, structures representing many of the tissues of the body were found.

Now, the one structure which is present in all the embryomata is that which is derived from the ectodermal layer, the epidermis. Wilms ascribes this to the fact that the ectodermal layer is developed first, and, in fact, may be the only layer which appears in some of these tumours. Now, in the ovarian dermoids at any rate, it is usual for all three layers—epiblast, mesoblast, and hypoblast—to be represented, but the portions which are developed from the two inner layers may be limited because of the pressure exercised on them by the surrounding structures or by their own growth, or because of other abnormal conditions under which they exist. In some cases they break down and disappear owing to a hæmorrhage into their substance or to interference with their nutrition.

Bostroem (2), in describing these tumours, has stated that intermediate grades exist in the complexity of the growths, from the very simple to the embryomata. The simplest, such as my first case with just the layer of epithelium, would be called by him a cholesteatoma. The complexity of the series increases by the addition of dermal and other structures, until at last we find growths which contain portions of the skeleton and representatives of many of the tissues of the body.

Other characteristics which, according to Bostroem, are shared by all of them, are:—

1. They are all intimately connected with the pia mater, and the active cells from which the tumour spreads are situated in that membrane.
2. They are always found in the basal portions of the brain, and in or near the middle line.
3. They all start in the early stages of embryonal life, *i.e.* between the third and fifth week of development.
4. They grow very slowly, and rarely give rise to any symptoms.

The gradual increase in the complexity of this series of tumours, together with these characteristics, which are common to them all, suggest that they must have a similar origin.

Hitherto, as Bostroem says, they have been ascribed to a penetration, at a very early period of embryonal development, of a series of ectodermal cells into that layer from which the

nervous system is developed; it is from these included cells that the various contents of the tumour are derived. These included cells must contain potentialities of the various tissues mentioned above, but their powers of development are limited by the abnormal conditions under which they exist.

No satisfactory explanation of such an inclusion of cells has yet been offered.

Lustig (3), speaking of the growth of these tumours in the sexual glands, says they can be logically explained by the theory of Weissmann, by admitting that in a germinal cell the reserve plasm, which is contained in these cells, has accidentally burst into activity, and has led to the formation of cells which are very similar to those found in a very early period of embryonic development, and that from these cells the various tissues contained in the tumour are derived. He does not offer any explanation of their occurrence in any other part of the body.

The subject was approached from an altogether different point of view by Wilms. In his comprehensive work on dermoid cysts and embryomata, without explaining all the steps by which these tumours have arisen in all parts of the body, he suggested the probability of their having sprung from "wandering germ cells."

Dealing with ovarian dermoid cysts, after showing that the theories which have been brought forward to explain their origin, viz. abnormal pregnancy, irritation or stimulation of some Nisus Formativus in the membrana granulosa, inclusio foetus in foetu, are untenable, he says that "dermoid cysts of the ovary can only spring from egg cells."

Again, when speaking of Beck's cases of tumours of various complexity—some containing portions of skeleton and of some of the organs of the body—in the pharyngeal and nasal cavities, he said that Beck could not decide whether they had sprung from wandering germ cells, or whether they owed their origin to a duplication of a series of cells.

Then, with regard to the tumours—ciliated epithelial cysts and dermoids—in the thorax, he said "they all may be traced to a scattering of germ cells in very early foetal life."

Lastly, when giving his conclusions on the origin of the teratomata, which grow in the nasal cavities, in the base of the brain, and in the cranial cavity, he said:—



1. The numerous branchial clefts of the head, the formation of the mouth, and the projection inwards of the hypophysis, offer many opportunities to the wandering germ cells.

2. Some of the teratomata may be produced by a duplication of a series of cells.

It is evident that although Wilms recognised that these tumours must have sprung from germ cells, *i.e.* from the only cells which are known to possess the potentialities necessary for such a growth, he made no attempt to explain the wandering of these cells. In the embryological works of Beard (4), however, we find a description of the earliest embryonal development in some of the fishes which throws much light on this phenomenon. In his investigations on the development of the *Raja batis*, Beard has shown that the earliest divisions of the fecundated ovum do not take part in the formation of an embryo, in fact the fecundated ovum passes through two series of divisions before any signs of an embryo appear.

The first series includes ten mitoses, and gives rise to a structure which Beard states corresponds to one stage in an alternation of generations, and leads up to the primitive germ cell. The next stage starts from this primitive germ cell, which divides into two, then into four, and so on through nine mitoses, giving rise to 512 primary germ cells.

All this has gone on without any appearance of an embryo. At this stage one of these 512 primary germ cells undertakes the formation of an embryo, and the other primary germ cells pass into this developing embryo along the yolk-stalk, which connects it with the yolk-sac.

This migration begins very early, even before the closure of the medullary plate, and lasts for only a short time.

It is possible to distinguish these primary germ cells, because when they are stained with osmic acid or Heidenhain's iron-hæmatoxylin, the yoke-plates which they contain are coloured a deep black, and they can thus be easily recognised amongst other cells.

These cells should find their way to the germinal nidus; but Beard has found in the *Raja batis* and in the *Pristiurus* that in their migration into the embryo some of them fail to find the germinal nidus, and they wander into other parts of the body. In the series of embryos of *Pristiurus* (5) examined, Beard

states "that no single embryo was devoid of germ cells in abnormal places." In the Raja batis they have been found in all the organs of the body except the thyroid and thymus glands.

Further investigations have led Beard to the conclusion that the capacity to form an embryo is not necessarily limited to one only of these primary germ cells, but that, for example in the skate, at least from 8 to 16 primary germ cells possess the potentialities necessary for such a development. In some animals, *e.g.* the armadillo, it seems to be a normal phenomenon to have more than one embryo developing within a single chorion.

If two of these cells, possessing the necessary potentialities, develop at the same time, the result will be that two embryos will be contained in a single chorion, and we shall have an example of what are known as "identical twins."

But if, instead of producing an embryo, a primary germ cell, endowed with such potentialities, wanders into the developing embryo and fails to reach the germinal nidus, it will stray into some other situation, and may there become encapsuled and remain inactive, or it may give rise to an incomplete development, owing to the abnormal conditions in which it is placed.

Such has, in all probability, been the sequence of events in my cases. In each of them a primary germ cell, endowed with the potentialities necessary for the development of an embryo, has migrated along the yolk-stalk, has failed to reach the germinal nidus, and has wandered to the frontal lobe of the brain, and there given rise to the tumours described above.

Now, although it is so rare to find these tumours in the human brain, it is by no means rare to discover wandering germ cells in the brains of fishes. They have frequently been found by Beard in the brain of the skate, and he has also discovered one on the spinal cord of a salamander.

The embryological researches of Beard therefore provide a basis of support for the opinion of Wilms that these embryomata spring from wandering germ cells, and we are thus enabled to obtain a clear view of the sequence of events which leads to the production of the embryomata, in place of the vague and unsatisfactory hypotheses which have been advanced to explain the origin of these tumours.

## REFERENCES.

1. Wilms. "Ueber die Dermoidcysten und Teratome, mit besonderer Berücksichtigung der Dermoides der Ovarien," *Deutsches Archiv für klinische Medizin*, Band lv.
2. Bostroem. "Ueber die pialen Epidermoide, Dermoides und Lipome, und duralen Dermoides," *Centralblatt für path. Anat.*, 1897, S. 1.
3. Lustig. *Patologia Generale*, 1901.
4. Beard. "A Morphological Continuity of Germ Cells as the Basis of Heredity and Variation," *Review of Neurology and Psychiatry*, 1904.
5. Beard. "The Germ Cells of Pristiurus," *Anatomischer Anzeiger*, Band xxi., 1902.

### THE RECOGNITION OF SEGMENTAL LEVELS IN THE CERVICAL AND LUMBAR ENLARGEMENTS OF THE SPINAL CORD FROM THE APPEARANCE OF THE TRANSVERSE SECTION.

By EDWIN BRAMWELL, M.B., F.R.C.P.E., M.R.C.P.Lond.

THE correlation of clinical symptoms present during life with anatomical lesions met with post-mortem necessitates in certain cases an exact determination, so far as possible, of the segmental level in the spinal cord from which any section under examination has been taken. This result may be arrived at by counting and marking the nerve roots and segments before the cord has been subdivided. Unless, however, the dura mater has been preserved intact so that it is possible to trace the roots from the point at which they penetrate this membrane to the point at which they join the spinal cord itself, the delimitation of the individual segments will be attended with considerable difficulty, particularly in the lumbo-sacral region.

When the segments have not been numbered and marked previous to subdivision of the spinal cord, the level of any section can only be gauged by distinctive characters which that section may present.

Dr Alexander Bruce, in his "Topographical Atlas of the Spinal Cord," has figured a series of very beautiful photogravures illustrating the special features of the transverse section at the level of each segment. The cord from which his figures are taken was the last and most perfect of five which were specially examined

for this purpose. The cord was divided into segments by a series of horizontal incisions passing through it immediately below the lowest fibres of each entering nerve root. Each segment was cut into serial sections, of which every tenth was stained in order to demonstrate the relative arrangement and proportions of the grey and white matter. "From the total number of sections in each segment a selection was made of one which appeared to be the most characteristic of that segment. Where the transit in form of grey to white matter in any one segment was so great that a single section could not be regarded as typical of that segment (as was the case in the eighth cervical segment), two sections were chosen." The sections are reproduced with a magnification of ten diameters. Other sections were stained to show the arrangement of the nerve cells, but since this subject is beside the purpose of the present communication, further reference thereto is unnecessary. Dr Bruce concluded from his research that although there are minor individual variations which appear to be mainly dependent upon differences in the shape of the cord, the "type of form" of the grey matter and the type of outline of the anterior cornu is constant for each segment or for corresponding parts of segments in different cords. He remarks, however, that it is very difficult, if not impossible, to identify individual segments in the dorsal region from the 3rd to the 10th inclusive, although it is possible to tell whether any given section belongs to the upper part, *i.e.* from the 3rd to the 6th, or to the lower, *i.e.* from the 7th to the 10th inclusive.

While engaged in cutting a normal cord for comparative purposes, it struck the writer that it would be interesting to examine a series of cords and to illustrate the appearances presented by sections taken from identical levels in order to show the amount of variation that might occur.

In the plate which accompanies this communication, outline drawings illustrating the shape and size of the transverse section of six spinal cords at successive segmental levels in the cervical and lumbo-sacral enlargements are figured. Before proceeding to refer to these appearances, it is necessary to describe the method which has been employed.

*Method.*—The six spinal cords, sections from which are figured on Plate 22, were removed from subjects free during life, so far as was known, from nervous disease. In each case

the cord was obtained from the level of the second or third cervical segment to the filum terminale, the dura mater being preserved intact. The dura was slit up from top to bottom both anteriorly and posteriorly and the cord was placed in 10 per cent. formalin. A few days later the roots were counted and a piece of silk attached to the first dorsal, first lumbar, and first sacral. *The method adopted in enumerating the roots was to regard the first small root caudal to the cervical enlargement as the second dorsal and to count up and down from this level.* The cord was then divided into segments by a series of transverse incisions made at the lowermost level of each entering root. Each segment was further subdivided into two equal parts by a transverse incision passing as nearly as possible through its centre. For the purposes of the present investigation the lower half of each segment was used. A horsehair was passed from above downwards through each piece of cord in the region of the left anterior horn. The lower halves of successive spinal segments were thus preserved in series. After hardening in Weigert's chrome alum solution and embedding in photoxylin in the usual way, each individual piece of cord was mounted on a numbered block, care being taken in each instance to see that its cephalic extremity was placed uppermost. Several sections were then taken from each block, and one or more of these were stained by Ford Robertson's modification of Heller's method and mounted. The sections from each segment were stained and mounted separately in order to avoid all risk of confusion, and with the same object in view each slide was numbered as the preparation was completed. In this way one or two sections were obtained from near the centre of every segment below the level of the second or third cervical. Careful camera lucida drawings of certain segments (C4 to D1 and L1 to S1) were then made. The drawings so obtained were photographed and are figured on the plate which illustrates this paper.

Before proceeding to refer to the features which characterise the different levels, two explanatory remarks are necessary. In the case of Cord I., for instance, there appeared to be 13 dorsal segments, if the first small root caudal to the cervical enlargement was regarded as D2. It follows that had this method of enumeration been alone adopted in the case of this cord, the lumbar segments would have all read one lower than is

figured on the plate. On counting the roots from below upwards, however, the lumbar sections were found to be correctly numbered as here depicted. It would seem, then, that in the first cord there was an extra dorsal root, or, what is perhaps more likely, that the whole cervical enlargement was displaced one segment upwards. For purposes of comparison it was considered advisable in numbering the segments in the case of this cord to omit one of the dorsal segments.

A further point to which the attention of the reader is directed is the transposition of the sections C5 and C6 of Cord VI., a mistake which was only discovered on examining the blocks after the drawings had been photographed and the plate prepared.

*Remarks.*—The result of this investigation has been to confirm Dr Bruce's conclusion that there is a type of outline of the anterior cornu which is characteristic of each segment in the cervical and lumbar enlargements. Let us now briefly point out the more prominent features which distinguish the transverse section each of the levels as represented in the plate.

*Cervical 4.*—The large size of this section as compared with the upper cervical segments is characteristic. The anterior horn is considerably broader than in the segments above this level. The anterior cornua in sections at this level and at the level of first lumbar segment are very similar in shape. The size of the section, the broader posterior horns of the first lumbar, and the projection into the posterior columns which is produced by Clarke's group in the upper lumbar region are, however, points by which these sections can be readily distinguished. (Cerv. 4, Cord III.)<sup>1</sup>

*Cervical 5.*—The prominent pointed antero-lateral angle of the anterior horn is the distinctive feature of this level. The postero-lateral angle is also pointed—a feature in which this segment differs from the eighth segment—with sections from the upper part of which (compare Cord V., Cervical 8) it might perhaps be confounded. (Cerv. 5, Cord I.)

*Cervical 6.*—The antero-lateral angle is not nearly so promi-

<sup>1</sup> The reference in brackets indicates the figure in the plate which most closely corresponds to the section depicted by Dr Bruce in his Atlas as typical of this segment. In this work the appearances of the transverse section are described in greater detail than in the present communication.

nent as in the preceding segment. A second antero-lateral angle, as pointed out by Dr Bruce, is characteristic of this level. This is especially well seen in the sections of Cords V. and VI. Note once more that Cervical 5 and Cervical 6 of Cord VI. have been transposed. (Cerv. 6, Cord V.)

*Cervical 7.*—The square or rhomboidal shape of the anterior horn distinguishes this level. (Cerv. 7, Cord I.)

*Cervical 8.*—The concave anterior border is distinctive. In the upper part of the segment an antero-lateral angle is figured by Dr Bruce as characteristic (compare Cerv. 8, Cord V.). This section, however, may be distinguished from the fifth cervical, to which it bears some resemblance, by its rounded postero-lateral angle. (Cerv. 8, Cord V. = upper part, Cerv. 8, Cord IV. = lower part.)

*Dorsal 1.*—The triangular, somewhat boomerang shape of the anterior horn is a feature by which this segment may be recognised without difficulty. In the section from Cord I. the anterior horn approaches in shape the type met with at the level of the second dorsal. (Dorsal 1, Cord IV.)

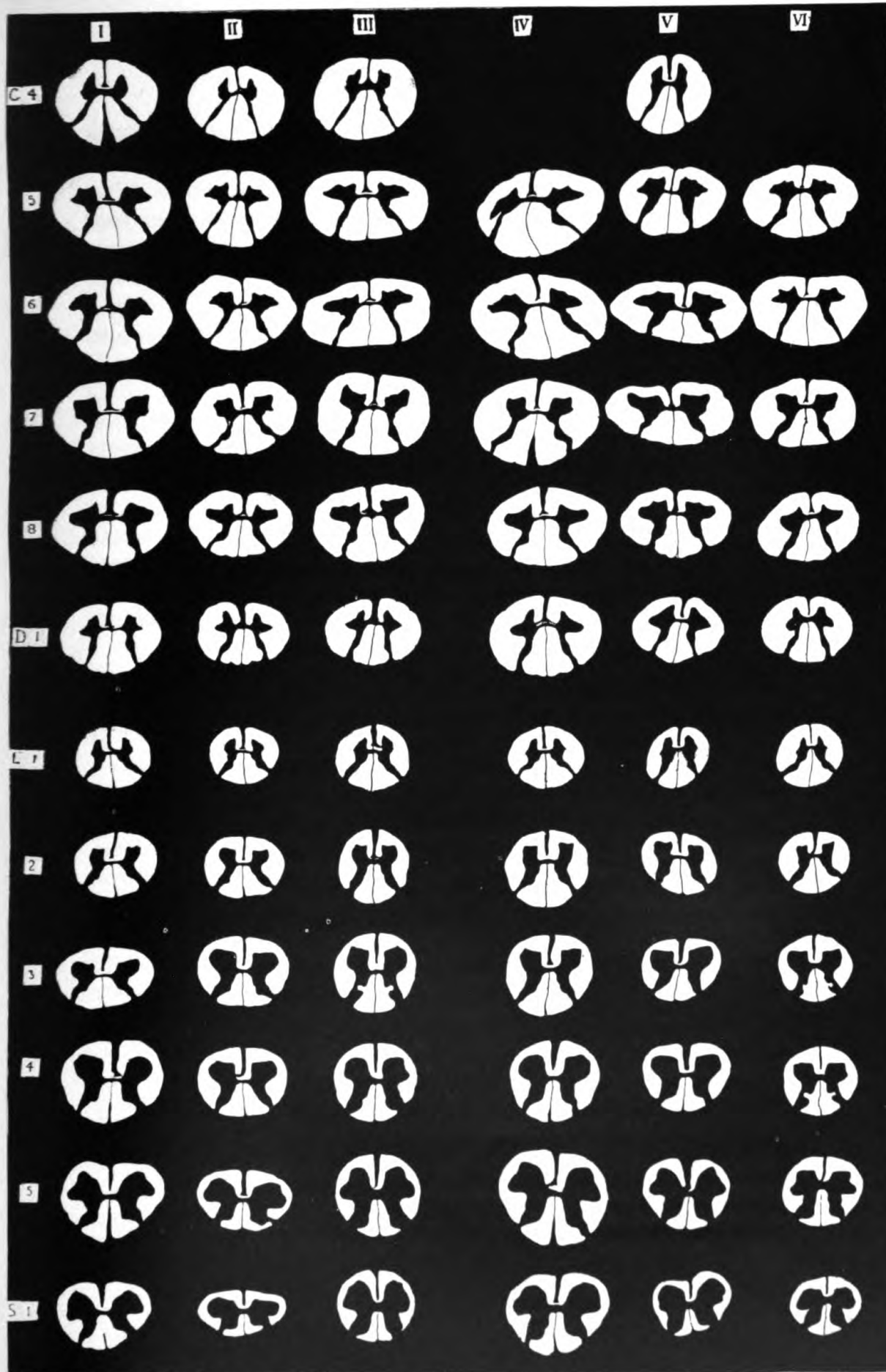
*Lumbar 1.*—At this level, as has been already pointed out, the anterior horn bears a considerable resemblance in shape to the fourth cervical segment, from which the section may be distinguished by its smaller size, by the greater relative breadth of the posterior horn, and by the prominent projection which is formed by Clarke's column. As in the case of the fourth cervical segment, the first lumbar is to be distinguished from the segments immediately above it by the greater breadth of the anterior horn. (Lumb. 1, Cord III.)

*Lumbar 2.*—The grey matter here assumes the shape of a narrow goblet. Dr Bruce's representation corresponds more closely to Cord V., Lumbar 3, than to any of the sections here figured from this level. (Lumb. 3, Cord V.)

*Lumbar 3.*—The grey matter still retains a goblet shape, although the cup is one of considerably greater capacity. (Lumb. 4, Cord V.)

*Lumbar 4.*—The rounded or hood-shaped anterior horn, as Dr Bruce describes it, is the striking feature of sections at this level. (Lumb. 4, Cord III.)

*Lumbar 5.*—The prominent antero-lateral and postero-lateral projections give the anterior horn at this level a resem-







blance to a hatchet with a concave cutting edge. (Lumb. 5, Cord I.)

*Sacral 1.*—The hatchet shape is still seen at this level, but the cutting edge of the hatchet is now convex. This section is somewhat similar to the fourth lumbar, from which it may be distinguished, however, by the acute angle formed by the white matter of the lateral columns as compared with the obtuse angle in the case of the fourth lumbar. (Sac. 1, Cord IV.)

---

## Abstracts

### ANATOMY.

**DISTRIBUTION OF THE CELLS IN THE INTERMEDIO-  
(162) LATERAL TRACT OF THE SPINAL CORD.** ALEXANDER  
BRUCE, *Trans. Roy. Soc. Edin.*, Vol. xlv., Part 1, 1906.

In a research on the intermedio-lateral tract in the cervico-dorsolumbar region of the cord, the author comes to the following conclusions :—

1. The intermedio-lateral tract may be defined as a tract composed of a special series of nerve-cells, situated at the outer margin of that portion of the grey matter which lies between the anterior and posterior cornua. These cells are not necessarily limited to the lateral cornua.

2. Within the spinal cord the tract is found in three regions : (1) in the upper cervical region as low as C. 4; (2) in the lower cervical, the dorsal, and the upper lumbar regions; and (3) in the lower sacral region (below the lower part of the third sacral segment).

3. It is absent in the cervical enlargement from C. 5 to C. 7 inclusive, and in the lumbo-sacral region from L. 3 to the upper part of S. 3 inclusive.

4. In that portion of the tract which is at present under consideration—viz. the second of the above-mentioned divisions—its component cells are found mainly in two positions: (*a*) in the lateral horn proper, or in analogous positions above the level at which the lateral horn is fully constituted; and (*b*) along the margin of that part of the grey matter which is in immediate relationship to the *formatio reticularis*, and also among the strands of the *formatio reticularis* itself. For convenience of description

and reference these may be distinguished as the *apical cells* and the *reticular cells*.

5. The apical and reticular cell-systems have not a co-extensive longitudinal distribution.

6. The apical cells are found between the middle of the upper half of the eighth cervical segment and the lower end of the second lumbar, or the extreme upper part of the third lumbar segment.

7. The reticular cells are first met with in the lower half of the second dorsal segment, and have the same lower limit as the apical series. They are not present in the cervical enlargement.

8. The upper part of the apical cell-series is composed of cells which are either situated in the white matter at some little distance behind the lateral part of the anterior horn, or are applied more or less closely to the grey matter. In all cases the cells are distinct from the large motor cells in their position, size, form, and grouping. No transitional forms are anywhere found between the cells of the two series.

9. The lateral horn is not fully constituted above the lower half of the first dorsal segment. This horn is not a transition from the lateral part of the anterior horn, but is a new and independent formation. It is represented in C. 8 and the upper part of D. 1 by the outlying cells of the intermedio-lateral tract.

10. The lateral horns of the two sides may show a want of symmetry in size and form, notably in the lower dorsal and lumbar regions. In the tenth, eleventh, and twelfth dorsal segments the apical cells lay in a plane posterior to the central canal.

11. The middle cells described by Waldeyer do not form any part of the intermedio-lateral tract.

12. The cells of the intermedio-lateral tract vary in size from 12  $\mu$  to 60  $\mu$ .

13. The apical and reticular cells could not be distinguished by any essential difference in their form, size, or structure. Large and small cells lying in close juxta-position may be present in both series in any one section. It has not been found that any group is composed entirely of large or of small cells. Large cells were relatively more numerous towards the lower end of the tract, but they were present alike in the apical and in the reticular series.

14. The number of cells in the intermedio-lateral tract is vastly greater than has hitherto been recognised.

15. The cells of the intermedio-lateral tract do not form a continuous column, but occur throughout the tract in groups or clusters.

16. These groups are not symmetrical on the two sides, although they may present a general resemblance to each other. There appears to be a larger number of cells on the left side in the lower cervical and upper dorsal regions. In the tenth dorsal segment there is a large excess on the right side.

17. These groups or clusters vary in size and form and in their distance from each other.

18. In each segment the cell-groups are arranged in a manner which may be regarded as characteristic of that segment.

19. The number of groups in each segment is somewhat difficult to determine in some cases; generally the number is fairly equal on the two sides.

20. At the upper and lower extremities of the tract there is a tendency for the groups to appear suddenly, to rise rapidly to a maximum, and then quickly to disappear. Towards the centre of the tract—below the fifth and above the tenth dorsal segments—the groups are less separated from each other. They rise slowly, persist for a considerable length, and diminish slowly. In this region the maximum number of cells attained is never so great as towards the extremities of the tract.

21. There is a remarkable increase in the number of the cells in the third dorsal segment. There is a marked transition in the form of the groups in the middle of the fifth dorsal segment, and another at the middle of the ninth dorsal segment.

22. The intermedio-lateral tract has a vascular supply largely independent of that of the motor cells of the anterior cornu.

23. The segmentation of the tract into groups or clusters of cells is not due to the distribution of blood-vessels or of the root-fibres, but is probably in some way related to their function.

It is pointed out that the greatest outflow of the sympathetic fibres from the cord, as indicated by the researches of Gaskell and Langley, corresponds in a remarkable manner with the position of the greatest number of the cells in the intermedio-lateral tract. Reference is made to the researches of Anderson and Herring and of Onuf and Collins, with regard to the probability that the intermedio-lateral tract is the source of the fibres of the sympathetic system.

The paper is accompanied by a graphic chart of the distribution of the fibres, and by 24 figures illustrating the position and distribution of the cells.

AUTHOR'S ABSTRACT.

## PHYSIOLOGY.

### ON THE PREPARATION OF CHOLESTERIN FROM BRAIN.

(163) O. ROSENHEIM, *Journ. of Physiol.*, March 1906, p. 104.

**THE CHOLESTERIN OF THE BRAIN.** M. CHRISTINE TEBB, *Ibid.*, (164) p. 106.

In the first paper a modification of Bünz' method for the preparation of cholesterin is described.

By means of this method human brain substance was examined for the presence of cholesterin-ethers. The results given in the second paper confirm Bünz' statement, that cholesterin occurs as such in brain and not in the form of ethereal salts.

W. CRAMER.

### **PATHOLOGY.**

**THE SUPRARENAL CAPSULES IN CASES OF NERVOUS AND (165) OTHER DISEASES.** F. W. MOTT and W. D. HALLIBURTON, *Journ. of Physiol.*, March 13, 1906.

IN this précis of their paper read before the Physiological Society on January 20, 1906, the authors state that having noticed the frequency with which atrophy or disintegration of the suprarenal capsules occurred at autopsies on cases which had died in the London County Asylums, and looking to the very special relationship between the nervous system and the medulla of the suprarenal, they examined the suprarenals in over seventy successive cases in order to determine if there was any connection between nervous disease and suprarenal atrophy.

The glands were examined histologically, by Vulpian's colour reaction with ferric chloride, and also physiologically for adrenaline.

In half the cases the glands were atrophied, degenerated, or disintegrated, but no connection could be traced between such a condition and the original nervous or mental disease for which the patient had been admitted to the asylum.

Suprarenal atrophy appeared, however, to be related to the secondary diseases from which the patient ultimately died, in the majority of cases where it occurred the patient having suffered from chronic disease.

The authors state that the observations suggest the possibility that the fatal termination of chronic diseases may be accelerated by the lack of suprarenal secretion, and consequent circulatory depression.

A. DINGWALL-FORDYCE.

**POST-MORTEM CHANGES IN THE NEUROFIBRILS.** (Alterations (166) *cadaveriques des neurofibrilles*.) LACHE, *Rev. Neurol.*, No. 5, 1906.

IN this paper the author gives the results of an investigation into the post-mortem changes of neurofibrils in the nerve cell and in the peripheral nerve. In the large motor nerve cells the peri-

nuclear fibrils are first attacked, and the process gradually spreads to the dendrites. The change consists of a granular degeneration, which leads to a complete disappearance of the fibrils. But with a high magnification it is seen that the fibrils in the dendrites are also altered, but the granules are larger and stain more darkly.

In the sensory cells, in the cells of Purkinje, and in the cells of the polymorphous layer of the cortex, all the fibrils become affected at the same time. In both cases the degeneration goes on until the cell is represented by a round mass of amorphous protoplasm, in which lie a few dark grains, or there may remain only the nucleus surrounded by a thin layer of homogeneous cytoplasm. The fibrils of the peripheral nerves undergo a process of fragmentation and decolorisation. In the spinal cord the granular disintegration of the large fibres is very slow.

Finally the author draws attention to two facts, first, that the changes which take place after death resemble those which occur when the cell is attacked by a poison during the life of the organism, and pass through the same stages, viz. alteration of the protoplasm, then of the nucleus, and lastly, of the nucleolus, and secondly, that the changes begin in that portion of the cell which is developed latest.

There is, therefore, no essential difference between the granular degeneration of the intracellular neurofibrils in the cadaver and the regressive changes of the fibrils when attacked by a toxin.

R. G. ROWS.

**ON THE DETECTION OF RHIZOPODS IN TWO CASES OF (167) ACUTE ANTERIOR POLIOMYELITIS.** (Ueber den Befund von Rhizopoden bei zwei Fällen von Poliomyelitis acuta.)  
V. ELLERMANN, *Centralbl. f. Bakt.*, etc., 1. Abt. Originale, 1906, Bd. xl., H. 5.

In the fluid obtained from two cases of poliomyelitis anterior acuta by means of lumbar puncture, and with stringent precautions so as to prevent contamination, the author found bodies which he regards as rhizopods. When fresh preparations of the fluid were examined on a warm stage, the parasites presented active amoeboid movement. In films stained by Leishman's method, the parasites were 10-15 $\mu$  in diameter; each contained a small ring-shaped nucleus; the cytoplasm was not granular; the pseudopodia were numerous and terminated in long, slender filopodia. Some of the parasites appeared to be phagocytic towards lymphocytes, but not towards erythrocytes. The appearances of the parasites are shown in two illustrations.

W. T. RITCHIE.

**MENINGOCOCCAL PHARYNGITIS AS A CAUSAL FACTOR OF**

(168) **EPIDEMIC CEREBRO-SPINAL MENINGITIS.** (*Die Meningococcenpharyngitis als Grundlage der epidemischen Genickstarre.*) A. OSTERMANN, *Deutsche med. Wchnschr.*, March 15, 1906, p. 414.

**MENINGOCOCCI** have not hitherto been often isolated from the nose or naso-pharynx of healthy individuals. Albrecht and Ghon, examining 15 persons, found meningococci in only one instance, a father whose child had died of meningitis. Weichselbaum and Ghon have examined the naso-pharyngeal secretion from a large number of healthy persons during the recent epidemic of cerebro-spinal meningitis in Silesia, but in only three instances did they detect meningococci; whilst v. Lingelsheim found those cocci in 24 out of 346 healthy persons who were in contact with cases of that disease.

The author instituted bacteriological investigations on the upper respiratory tract of the members of six families, in which there were children suffering from cerebro-spinal meningitis. He found meningococci in members of each of these families. Of 24 persons examined, the naso-pharynx of 17 harboured cocci, which were proved by agglutinating sera to be meningococci. Several of these persons presented naso-pharyngeal catarrh, but usually without fever, headache, or other sign of constitutional disturbance. Such persons affected with meningococcal pharyngitis constitute the chief foci of infection. The upper part of the naso-pharynx is the favourite site of the meningococci, which were less frequently detected in the nose or on the tonsils. At a time when there was no epidemic, the author examined 50 school children and 10 adults, without once finding meningococci in the naso-pharynx.

The chief prophylactic measures to be adopted when epidemic cerebro-spinal meningitis is prevalent are the closing of schools, or the detention from school of children living in a house where there is a case of that disease; the disinfection of sputum, of handkerchiefs, and of the patient's room; and the instruction of the laity as to the manner in which the infection is propagated.

W. T. RITCHIE.

**THE PATHOLOGY OF EPILEPSY.** JOHN TURNER, *Brit. Med. Journal*, March 3, 1906, p. 496.

THE author holds that epilepsy is a disease occurring in persons with a defectively developed nervous system, associated with a morbid condition of the blood, whereby it shows a special tendency to intravascular clotting; and that the immediate cause of the

fits is sudden stasis of the blood stream resulting from the blocking of cerebral vessels by the intravascular clots.

As regards the features pointing to a defectively developed nervous system, and which may be regarded as stigmata of degeneration, Dr Turner mentions the two following characteristics: (a) A variety of nerve cell which, as Lugaro, Bevan Lewis, and others have pointed out, represents an embryonic form, and is a common feature of the brains of imbeciles. This type of cell is most clearly seen in the Betz cells of the ascending frontal gyrus, and was observed by the author in 77 per cent. of his cases of epilepsy. (b) The persistence of subcortical nerve cells, a character first noted by Roncoroné. These cells exist in large numbers throughout life in the lower animals, but although found in infants they soon, with the growth of the child, become less, and at adult age few remain. In imbeciles, on the other hand, they tend to persist, so that at all ages they are met with in large numbers. They are also commonly seen in the brains of epileptics. Dr Turner also describes cells in the brains of epileptics similar to what has been observed in the brains of dogs after ligature of the cerebral arteries. He lays stress upon this appearance as indicating a like condition in epilepsy, viz. stasis of the cerebral circulation.

The characteristic vascular changes which he has observed consist of hyaline masses, spheres, finely granular clots and fibrine threads occupying the lumen of the smaller cerebral blood-vessels, the walls of which, however, present a normal appearance. These clots he ascribes to the amalgamation of blood plates, as their reaction to staining reagents indicates the presence of phosphorus, and points to the nucleo-proteid character of the clot. That the clots are not post-mortem changes, is clearly shown by the fact that proximal to these obstructions are often seen rupture of the blood-vessel wall and effusion of blood into the perivascular space.

The author ascribes the sclerosis of the cornu ammonis, so well known a feature of epileptic brains, to the deprivation of the normal blood supply to this part, as a result of complete or partial thrombosis of its nutrient vessels, whereby the tissues are, as it were, starved.

The cornu ammonis is, however, not the only part of the brain affected by this sclerotic change, for similar features were seen in the occipital lobe and in the cerebellum.

In support of his contention that the convulsions in epilepsy are due to the deprivation from the cortical areas of arterial blood in consequence of the thrombotic obstructions which he has observed, Dr Turner refers to the well-known Kussmaul and Penner experiments, in which general convulsions were produced in rabbits after ligature of the subclavian and innominate arteries;



and to an observation of L. Hill's, who produced spasm in himself by compressing one of his carotid arteries; this author also states that sudden occlusion of one carotid artery may in some persons produce a march of epileptic spasm preceded by an aura.

W. ALDEEN TURNER.

### CLINICAL NEUROLOGY.

#### POLYNEURITIS AS A SEQUELA OF GERMAN MEASLES.

(170) (*Polynévrite suite de rubéole.*) REVILLIOD and LONG,  
*Arch. de méd. des enfants*, March 1906.

SEQUELÆ of any kind in this usually trivial exanthem are so rare, that the following case reported by Revilliod and Long is of considerable interest. A boy, aged eight, had a mild attack of German measles without any rise of temperature or constitutional disturbance. The eruption completely disappeared in forty-eight hours. Ten days later paralytic phenomena developed, which were at first confined to the lower extremities. His walk became uncertain and staggering, and he soon found it impossible to go upstairs or downstairs, to get into bed without assistance, or even to rise from the dorsal decubitus into a sitting posture. The trunk and upper limbs soon became involved, and neuritic pains simultaneously developed. For three weeks the motor impairment became worse and then began to improve, but the normal condition was not regained till the end of two months. The pains disappeared long before the return of motility. The tendon reflexes were lost throughout the affection and remained so long after apparent recovery. There was no definite muscular atrophy nor reaction of degeneration. Contrary to what is usually found in motor polyneuritis, paralysis of the trunk muscles was much more accentuated than that of the limbs, while in the extremities the proximal muscles were more affected than the distal ones. The neuritic pains were also more marked at the root of the limbs. Diplopia, a rare manifestation of polyneuritis, was present for some days. The child had been kept under careful observation throughout his illness, and the writers confidently assert that all concomitant infectious diseases, in particular diphtheria, could be excluded from the causation of the polyneuritis.

J. D. ROLLESTON.

#### TABES, GENERAL PARALYSIS, AND SYPHILIS. (*Zur Tabes-*

(171) *Paralyse-Syphilis-Frage.*) ARTH. HERM. HÜBNER, *Neurolog. Centralbl.*, March 16, 1906, p. 242, I. Beitrag.

THE author first refers to the rate at which the opponents of the Erb-Fournier hypothesis have diminished in the past few

years, and mentions the historical account of the subject given in "Nonne's Syphilis und Nervensystem," 1902, and Erb, *Berliner klin. Woch.*, 1904. Hübner's present researches deal with an inquiry into these diseases amongst the women of the lower classes in Berlin. He has had special opportunities in investigating the police and club statistics of such women. The alleged infrequency of parasyphilitic diseases among prostitutes is to be accounted for partly by the loss in professional capacity sustained by those who acquire syphilis and its consequences—thus diminishing their apparent numbers through exclusion from the ranks—and partly by the extraordinary high numbers in statistics, such as Glaser's of women who had suffered from syphilis so recently as to make parasyphilis as yet impossible. For instance, if the number of women infected within five years are excluded from Glaser's statistics, the percentage of tabetics amongst those remaining is more than doubled. Hübner could find no reliable published investigations of general paralysis among prostitutes. He has made notes in hospital work of 97 prostitutes over the age of 25. Of those living, 20·9% had general paralysis, 7·0% tabes and 85% cerebro-spinal syphilis. This total of 32·9% is twice the percentage seen in other women under similar circumstances. The proportion of tabes and cerebro-spinal syphilis is 4 times that given by Glaser and 5·7 that given by Mendel, as these authors have not allowed for the fallacies above mentioned. In the 41 cases which had been examined post-mortem, excluding those dying of accident, 58·5% had general paralysis, 5% tabes, and 24·4% cerebro-spinal syphilis, or a total of 87·9%. By the side of these are the figures of the 150 women, other than prostitutes, that were examined post-mortem in the last two years, showing a frequency of general paralysis 16·7%, tabes 2·7%, cerebro-spinal syphilis 4%, or a total of 23·4%. In 1903, 0·3% of the men dying in Berlin were certified to have tabes, and 0·17% of the women. On adding the cases of 55 living and 38 dead prostitutes to his own list, cases obtained from the Rummelsburg workhouse, he reaches the conclusion that 42·8% of all prostitutes suffer from parasyphilis.

The next point dealt with is the comparative frequency of child-rearing in healthy women and in those with tabo-paralysis. Of 70 cases of general paralysis and 120 of other psychoses, 45·7% of the former were sterile and 14·4% of the latter, 5·7% of the former had only abortions compared with 2·5 of the latter, and 31·4% of the former had only full-time children in contrast with 74% of the latter. The paper concludes with several instructive examples of syphilitic families and refers to the importance of syphilis in other conditions, such as deafness, idiocy and defective speech.

ERNEST JONES.

**SOME FORMS OF TABES SELDOM DESCRIBED.** (Einige wenig (172) beschriebene Formen der Tabes dorsalis.) MICHAEL LAPINSKY, *Deutsch. Zeit. f. Nervenheilk.*, Bd. xxx., H. 3 u. 4, 1906, S. 178.

AFTER referring to the classical picture of tabes, one that is filled in chiefly by afferent phenomena, the author describes at length five cases that made their appearance with motor signs. The first of these was a girl of 23, whose father had suffered from syphilis; her initial symptom was a paralysis of the left leg, followed a week later by a paresis of the right hand. Subsequently indubitable signs of tabes developed. The second case, a man of 37, began with weakness of both legs and for some time it was impossible to be sure that the condition was not one of peripheral neuritis. The third case, an officer of 28, had suffered two years previously—seven after infection—with a temporary squint. Weakness appeared in both arms and legs, so that he could no longer ride or carry out his exercises, but it was accompanied by pain only after some months. Rombergism was, however, present early. The fourth, a woman of 45, suffered first from weakness of the lower limbs, the right, which had wasted  $\frac{3}{4}$  inches, being worse than the left. The tendon reflexes were slightly diminished, electrical reactions normal. The last case, a woman of 30, was very similar to the fourth. In all five unequivocal signs of tabes developed.

In reviewing the cases the author points out how misleading it is when tabes begins with muscular weakness, either of special groups or widespread, and the likelihood of the condition being mistaken for a complaint of the peripheral nerves, bony system, and especially of the joints. It was easy to eliminate functional disease and affections of the pyramidal tract or of the muscles themselves. Unfortunately, all the characteristic features of ataxy, as described by Oppenheim, may fail, as in these cases. Anterior horn cell affection may be excluded by the variability of the symptoms, and by their tendency in general to get better. However, the author holds it probable that slight chromolytic changes are present in these cells, analogous to those found after section of the posterior nerve roots in such experiments as those of Mott and Sherrington. The other possibility is that the tonus of the anterior horn cells is diminished, through the lessened sources of excitation which reach the spinal cord. Similar cases to those now described are referred to in the writings of Grasset and Frenkel. The latter mentions a case in which the motor symptoms had a quite acute onset, but were shown later to be part of a tabetic affection.

ERNEST JONES.

**SYMPTOMS AND TREATMENT OF EXTRA-MEDULLARY**

(173) **SPINAL NEW GROWTHS.** (Zur Symptomatology u. Therapie der sich im Umkreis des Rückenmarks entwickelnden Neubildungen.) H. OPPENHEIM, *Mittel. aus den Grenzgebieten der Med. u. Chir.*, 1906, H. 5, p. 607.

THE author records in this paper six cases of extra-medullary tumours at different levels. The symptoms varied considerably, but, as a rule, there was an early irritative stage, followed gradually by paralytic and spastic phenomena, but no marked extension of the process upwards or downwards accompanying the signs of increasing local compression. A typical Brown-Séquard syndrome developed when the tumour lay laterally to the cord. He finds also that the level of the tumour may be diagnosed too high when there is a concomitant meningeal affection. With regard to differential diagnosis, there is an affection which produces a picture practically indistinguishable from that of tumour, viz. a localised spinal meningeal hydropsy. Such fluid collections may result from narrowing of the spinal canal by caries, or from thickening of the meninges, and their influence is said even to be occasionally, in some unexplained manner, quite unilateral. The result of operative interference in these cases was—one complete recovery (operation eight years ago); one recovery with paresis from section of nerve roots; four deaths within a week of the operation.

J. H. HARVEY PIRIE.

**THE PRINCIPAL FORMS OF NERVOUS TROUBLES IN POTT'S**

(174) **DISEASE WITHOUT DEFORMITY.** (Les principales formes des troubles nerveux dans le mal de Pott sans gibbosité.) L. ALQUIER, *Nouv. Icon. de la Salpêtr.*, No. 1, 1906, p. 2.

IN grown-up people neuralgic pains almost always, and graver nervous symptoms frequently, precede by many months the first sign of spinal deformity. Such symptoms are therefore of the greatest importance as regards early diagnosis of vertebral caries. Much difficulty may be experienced in distinguishing between the effects of involvement of nerve roots, and of the cord itself, but clinically a combination of both is most common. For want of a better, the writer adopts a classification depending on the anatomical position of the disease.

1. Cases with signs of compression of the dorsal and upper lumbar cord and roots. These show early intermittent girdle pains, followed by numbness, tingling, and neuralgic pains of the legs. Next comes spastic paraplegia, gradual in onset or sudden, complete or partial, equal in both legs or more marked in one.

This may be accompanied by sensory disturbance—hyperæsthesia or anæsthesia of the whole body below the lesion, limited above by a band of girdle anæsthesia. The legs swell, sores develop, and in most cases there is retention of urine.

2. Cases with root and medullary compression of the filum terminale and cauda equina. Diagnosis in this group is extremely difficult unless there is obvious disease of the lumbo-sacral region. In every case there is early and severe pain in the legs, usually resembling sciatica and sometimes unilateral. Next appears flaccid paraplegia with muscular atrophy and incontinence of urine. Anæsthesia corresponding to the distribution of the lumbar and sacral roots may follow.

3. Cases with compression of the cervical cord and roots. Here also pains from pressure on the nerve roots form the first symptoms. Anæsthesia in the arms, usually of the dissociated variety, is next noted. Sooner or later flaccid paralysis with muscular atrophy ensues; and when pressure on the cord develops this is associated with spastic paralysis of the legs. A certain degree of ataxia is common. Bowel and bladder troubles are slight. When the disease is situated high up, symptoms due to involvement of the medulla are added. Special nerves may be implicated, *e.g.* the sympathetic giving rise to disturbances of the pupil, etc., and the hypoglossal resulting in partial or complete atrophy of the tongue.

Cases are detailed illustrating the above three groups. The differential diagnosis is next considered. In neuralgia of doubtful origin some help may be obtained from the injection of a local anæsthetic, when more relief will be obtained in a peripheral than in a root lesion. The result of a lumbar puncture is negative. The pain of Pott's disease is aggravated by walking and movement, and relieved by rest. Flaccid paralysis of the legs may simulate a multiple neuritis or a myopathy. Spastic paralysis of the legs may closely resemble a transverse myelitis, especially syphilitic. In cervical disease, with atrophy and dissociated anæsthesia of the arms, spasticity of the legs, and slight scoliosis, a diagnosis from syringomyelia is often difficult. Positive diagnostic signs are, firstly, those common to tuberculosis anywhere, and a reaction to tuberculin may be of help, although there are obvious fallacies connected therewith; and, secondly, those indicating vertebral disease, such as rigidity of the spine and pain on percussion. Skiagraphy gives comparatively little assistance. Diagnosis may sometimes be absolutely impossible, but every case should be investigated with the greatest care on account of the importance of early treatment.

The article is excellently illustrated with drawings and photographs.

HENRY J. DUNBAR.

**SPONDYLITIS DEFORMANS.** (*La Spondylose Rhigomélique ; ana-*  
(175) *tomie pathologique et pathogénie.*) PIERRE MARIE et ANDRÉ  
LÉRI, *Nouv. Icon. de la Salpêtr.*, No. 1, 1906, p. 32.

THIS disease differs essentially from chronic rheumatic, traumatic, and other conditions leading to ankylosis, and must be regarded as a definite, and, moreover, a comparatively common pathological entity. The cause in most cases is some infective agent, frequently gonorrhœal or tuberculous ; but constitutional predisposition and the effects of chill must also be taken into account. In 1899, Léri advanced the theory that the disease was of the nature of an ossification of the ligamentous structures of the joints affected, and that this was accompanied or preceded by a rarification of osseous tissue to which the condition was probably secondary or compensatory. The spinal column, the shoulders, and the hips are the usual seats of the disease; occasionally the sterno-clavicular joint and the knee ; and only rarely, and of late occurrence, the other joints of the extremities. The spine is curved antero-posteriorly, and the ligaments on the convexity show the most marked ossification. The curve is greatest in the cervico-dorsal region, because of the weight of the head. The thorax is always more or less flattened, and frequently also the pelvis. The femora may be ankylosed either in flexion or extension. In the early stages pain is constant. In the present article the writers record a case recently observed by them, and give a full account of the post-mortem examination, which fully confirms their previously expressed views as to the pathology of the disease. The article is illustrated by excellent photographs and diagrams.

HENRY J. DUNBAR.

**REPORT ON EPIDEMIC CEREBRO-SPINAL MENINGITIS IN**  
(176) **INDIA.** C. J. ROBERTSON-MILNE. Pp. 67. Government Print-  
ing Office, Calcutta, 1906.

THIS report considers the Historical, Clinical, Bacteriological, and Epidemiological aspects of the subject. Although the fact seems to have been generally overlooked, Cerebro-Spinal Fever, in both its epidemic and sporadic forms, has been recognised in India for almost a quarter of a century, and the records of the disease show that it is in every way identical with the malady as it has been observed in other countries. An outbreak in Persia about 1874-5 appears to be the only other recorded occurrence of the disease in Asia. In India it has most frequently attacked prisoners in jails : in some of these institutions the disease has prevailed irregularly

for prolonged periods, 593 cases (with 450 deaths) having occurred from 1881-1901 in a population averaging annually 100,123. No outbreak of any magnitude has been chronicled in the general community, although the disease has prevailed widely.

J. H. HARVEY PIRIE.

**THE FUNCTION OF THE LEFT PREFRONTAL LOBE.** CHARLES (177) PHELPS (New York), *Am. Journ. Med. Sciences*, March 1906.

FROM a series of 46 cases of left prefrontal involvement which Phelps reported in the *Am. Journ. of Med. Sciences* for May 1902, he came to the following conclusions: (a) "The more absolutely the lesion is limited to the left prefrontal lobe, the more positive and distinctive are the symptoms of mental default. (b) The integrity of the mental faculties remains unimpaired with right frontal lesion, even though it destroys the entire lobe or extends to the entire right hemisphere. (c) Exceptional instances in which seemingly opposite conditions exist are always reconcilable on more careful examination, with the assertion of an exclusive control of the mental faculties residing in the prefrontal region of the left side."

He now adds to the former series 11 more cases of similar nature, and concludes that 57 cases should form an adequate basis for a positive opinion upon the function of the left prefrontal lobe. In 3 of these cases the lesion was limited to the left lobe; in 3 more the right lobe was implicated, but to a lesser degree; in 5 the lesion was limited to the right lobe. He includes in his paper the history of each case, and the autopsy reports of fatal cases. His conclusions are that lesions limited to the posterior left frontal region occasion only motor defect; conjoint lesion of both prefrontal regions is always attended by mental decadence; lesion of the left prefrontal region alone is always attended by mental decadence; lesion of the right prefrontal region alone is never attended by mental decadence. He finds that the small number of cases which have been reported of left prefrontal lesions having unaffected minds and right prefrontal lesions with disordered minds are unworthy of credence. In such cases either no detailed history has been afforded, or the assertions have been found to be incompatible with the history as stated.

C. H. HOLMES.

**CEREBRAL MANIFESTATIONS OF HYPERTONUS IN SCLEROSED** (178) **ARTERIES.** WILLIAM RUSSELL, *Practitioner*, March 1906.

IN this paper the term "sclerosis," as applied to arteries, is confined to the uniform and widely distributed thickening of

arteries due to thickening of both the middle and internal coats. The condition is quite different from atheroma, although commonly confused with it. In arterio-sclerosis the arteries retain their power of contractility—may, indeed, be unduly sensitive to some of the impressions to which arteries respond. Whilst thickening of the arterial wall is easily recognised, it is always a question how much of it is due to permanent tissue increase, and how much to an excess of the normal tonic contraction of the muscular coat? It is to this increase of normal tonic contraction that the term *hypertonus* or *hypertonic contraction* is applied. As to the cause of hypertonic contraction, the general proposition may be laid down that deleterious substances of various kinds present in the circulation cause it. In this paper some of the cerebral symptoms due to hypertonus in sclerosed arteries are referred to. The first condition dealt with is the occurrence of hemiplegia, monoplegia, or aphasia without loss of consciousness. It is pointed out that the diagnosis lies usually between thrombosis and hypertonic contraction. The latter, as a cause of the conditions mentioned, has not been recognised, and yet it is probably more common than thrombosis. When the condition of the vessel wall in sclerosis is borne in mind, and when it is recognised that sclerosed vessels can become markedly contracted, so as to diminish or almost obliterate their lumen, it is evident that the condition can as effectually interfere with the nutrition of the portion of brain supplied by such arteries as if they were occluded by embolism or thrombosis. There is, however, this great practical difference, that, whereas the two latter conditions are permanent, the first-mentioned may be transitory, and can, to an important degree, be influenced by treatment. The degree of local spasm varies; in some instances it merely impoverishes the blood-supply of the part, and the symptoms are correspondingly slight. If, along with spasm-contraction of arteries, there is a feebly acting heart, the circulation in the part is necessarily still more reduced. Cases are given illustrating the foregoing. Other symptoms, due to this contraction of vessels, are pains in the head and attacks of giddiness. Another common type of case occurs, usually in old people, with all the characteristics, mental and vascular, which commonly accompany senility. These persons have attacks of restlessness, insomnia, and mental excitement. In these attacks it is usually found that there is a general hypertonic contraction of vessels, and that the symptoms pass off when the vascular condition is relieved.

AUTHOR'S ABSTRACT.



**HÆMORRHAGE FROM THE MIDDLE MENINGEAL ARTERY.**

(179) J. HOGARTH PRINGLE, *Scot. Med. and Surg. Journal*, Feb. 1906, p. 97.

THE author records fifteen cases of head injury followed by hæmorrhage from the middle meningeal artery which illustrate well the difficulties in making an accurate diagnosis and carrying out suitable treatment in such cases owing to the great variations in the symptoms produced by the extravasated blood.

Although all fifteen cases were associated with fracture of the skull, in two of them the tear of the artery occurred on the opposite side of the skull to the fracture, and were therefore comparable to the cases which have been recorded of tear of the artery without any fracture of the skull at all. Pringle's explanation of the mechanism in such cases is "that owing to the violence applied to the head the elasticity of the skull permits a rapid alteration in its shape, while the dura mater, less elastic than the bony case, does not react so quickly and gets lacerated in consequence."

In three of his cases the main trunk of the artery was injured, in five the anterior main branch, in three the posterior main branch, and in four several of the smaller branches.

In only one of the fifteen cases was the typical sequence of symptoms present, *i.e.* an initial period of unconsciousness due to concussion, followed by a lucid interval and finally by a return of unconsciousness with other symptoms of compression. In eight cases unconsciousness was present from the first and continued till death occurred. In only four of the cases was paralysis limited to the side of the body opposite to that of the head lesion. In nine cases the patients were able to move all their limbs spontaneously or after irritation. In six of the cases there was inequality of the pupils, and in all these the wider pupil was on the side of the hæmorrhage, while in five of them neither pupil reacted to light. In five cases the pupils were equal and active, in two they were equal but did not react to light.

In discussing the diagnosis, Pringle draws attention to the fact that the frequent combination of brain laceration, subarachnoid or subdural hæmorrhage, with extradural hæmorrhage, causes great difficulty. He inclines to the view that laceration and subarachnoid hæmorrhage more often cause general paralysis from the first, than extradural hæmorrhage. In two of his cases every symptom usually associated with middle meningeal hæmorrhage was absent till a very late stage. This was explained by a free escape of cerebro-spinal fluid from the interior of the skull. In one case the patient remained conscious for four days, till three hours before death, and in the second for twelve days after the injury. In both

cases the clot was over the posterior parietal region, and on the opposite side of the head to the fracture. Ten of the fifteen cases were operated on and four recovered. In the fatal cases subdural hæmorrhage and brain injury were present and were apparently the cause of death.

J. W. STRUTHERS.

**THE ONSET OF HEMIPLEGIA IN VASCULAR LESIONS. A**  
(180) ERNEST JONES, *Brain*, Autumn 1905, p. 527.

THIS paper—the first of a series—is based partly on a study of the autopsy records at University College Hospital for sixty-five years, and partly on a large number of published cases, the individual accounts of which have been studied by the author. The fallacies and advantages of this method of approach are considered. It is shewn that much of the clinical teaching on the subject is based on tradition and founded on insufficient evidence, and that there is great need of a large series of cases, the history of which has been critically studied and the diagnosis verified.

The main conclusions expressed are as follows:—

(1) Rest in bed, and to a greater extent sleep, protect to some extent against cerebral hæmorrhage. This seems to be a more accurate statement than that rest and sleep predispose to thrombosis; for, whereas only one-third of the number of hæmorrhage attacks that might have been expected to occur during sleep did so in fact, exactly the anticipated number of thrombosis attacks occurred then.

(2) The significance of severe exertion and of time of day has been over-estimated in the past.

(3) Consciousness is lost at the onset of half the cases of occluding lesions, and of three-quarters the cases of hæmorrhage. It is transitorily affected in many of the remaining cases. We have no accurate knowledge as to the depth or duration of the coma in the various lesions, or its relation to the completeness of the paralysis. Until these latter problems are decided, it is well to remember the danger of relying too much on this aid to diagnosis on account both of an important group of occluding lesions in which the onset is apoplectiform, and another group of hæmorrhage lesions in which the onset is quite insidious.

(4) The immediate prognosis is much graver when the onset is apoplectiform; especially is this so in cases of hæmorrhage. Contrariwise, late cases of hemiplegia due to hæmorrhage are less likely to have suffered from loss of consciousness, and are therefore the more likely to be attributed to thrombosis.

(5) Intraventricular hæmorrhage, which is nearly always secondary, may not cause loss of consciousness. On the other

hand, immediate loss of consciousness as the initial symptom may be due to extra-ventricular hæmorrhage.

(6) The immediate prognosis is much graver in cases of hæmorrhage than in cases of occluding lesions. The results of 828 cases of hæmorrhage, 158 of thrombosis, and 273 of embolism are as follows: over 30 per cent. of the first cases were fatal within 24 hours, half as many of the second, and a quarter as many of the third; nearly two-thirds of the first patients are dead in a week, and more than a third of the others. Of 20 cases of each lesion, 4 hæmorrhage ones would survive a month, 5 thrombosis ones and 9 embolism ones. Most of those that survive two years are cases of thrombosis.

(7) Of the cases in which blood is found in the ventricles, 60 per cent. die within 24 hours, and 90 per cent. in the first week. It is not very rare, however, for such cases to live a few weeks.

(8) On the first day the mortality in cases of hæmorrhage is heaviest amongst the younger men; in the next few weeks amongst the aged women.

(9) There is no indication that either hæmorrhage or thrombosis affects one side of the brain more often than the other. A right-sided lesion was present in 49·89 per cent. of 995 cases of the former, and 47·2 per cent. of 176 cases of the latter. Of 558 cases of embolism—three times the number in any previous collection—the lesion was right-sided in 40·48 per cent. Mathematically this means that a right-sided lesion in embolism cannot be much more common than a left one, that it may be either equally common or much less common. The usual statements to the last effect are unwarranted at present.

AUTHOR'S ABSTRACT.

**CLINICAL STUDY OF HEMIPLEGIA IN THE ADULT.** THEODORE  
(181) H. WEISENBURG, *Journal of the American Med. Assoc.*, Feb. 25,  
1905.

THIS paper is based on the study of 160 cases of hemiplegia, particular attention being paid in most of the cases to nine special points. The conclusions may be summarised as follows:—

1. *Heredity*.—In 14 out of 109 cases, at least one parent had suffered from hemiplegia; in one of these cases both parents had.

2. *Prehemiplegic Pain*.—Seventeen cases out of 109 suffered in this way. In 12 of these cases the pain occurred within 48 hours of the stroke; in the other cases at varying periods up to two years. They were mostly in the upper extremity, in the muscular tissues and joints. In five of the cases the pain ceased after the onset of the hemiplegia; in another seven it was mitigated.

3. *Posthemiplegic Pain*.—Of 109 cases, 27 had such pain, 12 had paræsthesia, 70 had soreness on the paralysed side; only five complained of no pain or ache. The largest group was to be correlated with contractures. Pain was commoner in cases where there were sensory changes.

4. *Respiration*.—In every one of the 160 cases the author confirmed Hughlings Jackson's observation that the upper part of the chest expands most on the paralysed side in quiet respiration, and least on that side in forced respiration. He points out, however, that in quiet respiration the chest retracts more on the sound side, which means that there must be more residual air left in the lung of the paralysed side. This would account for the greater expansion on that side, in order to take in an equal quantity of air on the two sides.

5. *Edema*.—In only two cases was this present to any extent. In one of them it was widespread on the paralysed side; in the other case, and in yet a third, a "succulent hand" only was present.

6. *Vasomotor Disturbances*.—Coldness is very common in the paralysed limbs. One patient did not perspire on the paralysed side, nor did he sun-tan on that side. Another patient had an eczematous eruption, limited to the paralysed side.

7. *Posthemiplegic Chorea*.—No case of choreiform tremor was observed, but a case with irregular tremor is described and its pathology discussed.

8. *Cerebral Muscular Atrophy*.—This was present in every case, but in very varying degree. It is always more marked in the upper limb, and usually most in the shoulder or hand muscles. The usual explanations are quoted.

9. *Arthropathies*.—One case of Charcot's painful joint was seen. On the other hand, the condition described by Marie was very common. In this there is limitation, and pain on passive movement, but no redness or swelling. The shoulder-joint is most often affected in these latter affections, and they are probably due to the forced immobility plus the pulling on the articulations by the weight of the paralysed member.

A. ERNEST JONES.

**SENSORY DISTURBANCES IN CEREBRAL HEMIPLEGIA.** (Über (182) die Sensibilitätsstörungen bei cerebralen Hemiplegien.) GEORG SANDBERG (Breslau), *Deut. Zeitschr. f. Nervenheilk.*, H. 3-4, 1906, p. 149.

OF 31 cases examined, the author found 22 with objective sensory disturbances. The majority corresponded to v. Strümpell's "posterior column type," *i.e.* there were disturbances of touch and deep

sensibility, while the pain sense and temperature sense were well preserved. In bulbar lesions all the sensory qualities may be affected. In five cases with only slight motor weakness there was marked stereognosis from disturbances of the pressure sense and sense of movement and position. J. H. HARVEY PIRIE.

**POST-HEMIPLEGIC MOVEMENTS.** (Beiträge zur Lehre der post-  
(183) hemiplegischen Bewegungstörungen.) ERNEST FREY, *Neurol.*  
*Centralbl.*, Dec. 1, 1905, p. 1104.

THE case under notice was one of right hemiplegia with contracture of the right lower face and upper extremity, the lower extremity being hypotonic. The left arm and leg were slightly spastic. Athetoid movements were observed in the right arm and occasionally in the left. The movements consisted of slow extension of the arm at the elbow, supination and pronation at the wrist, and flexion and extension of the fingers. The deep reflexes were brisk and there was slight ankle clonus on the right side. The pupils were of moderate size, the right being larger than the left, and both were inactive to light. The mental condition was one of apathetic dementia, and this prevented any satisfactory examination of the sensory system.

The case came to autopsy and after staining by Weigert-Waller's method, frontal sections at the proximal middle and distal ends of the lesion revealed the following lesions: (1) A somewhat large focus of hæmorrhage in the left optic thalamus which destroyed the external nucleus and the internal capsule bounding the thalamus at that spot. The fibres connecting the red nucleus to the thalamus and the lenticular nucleus were also destroyed. (2) Destruction of the middle and internal nuclei of the thalamus and of the upper part of the red nucleus. (3) Destruction of the middle and outer nuclei of the thalamus. There was slight degeneration of the fibres in the left crus, in the pons, and in the pyramidal tracts on the right side of the cord.

After discussing the various opinions which have been advanced as to the seat of lesions which may give rise to post-hemiplegic movements, the author draws the following conclusions: (1) That the post-hemiplegic movements were due to lesions of the thalamus or hypothalamic region. (2) That the thalamus is a co-ordination centre.

T. GRAINGER STEWART.

**A CASE OF SYRINGOBULBIA, &c.** (Un cas de Syringobulbie,  
(184) &c.) RAYMOND and GUILLAIN, *Rev. Neur.*, Jan. 30, 1906, p. 41.

THE interest of the case lies in the association of typical syringomyelic (cord) symptoms with others pointing as clearly to involve-

ment of the medulla. These were hemiatrophy of the muscles of the palate and of the pillars of the fauces on the right side, together with complete paralysis of the right vocal cord. These latter conditions constitute the syndrome of Avellis, so called, and signify paralysis of the internal branch of the vago-spinal nerve. The fact that in the present instance the facial nerve was intact would appear to corroborate the view that the vago-spinal and not the seventh is the principal nerve supply of the palate muscles.

S. A. K. WILSON.

**MULTIPLE LIPOMATA IN GENERAL PARALYSIS.** CONOLLY (185) NORMAN, *Journ. of Ment. Sc.*, Jan. 1906, p. 62.

THE patient was a male, aged 40, who presented well-marked speech trouble of the general paralytic character, general tremors, and tabetic symptoms. In addition to the usual mental symptoms of the disease, he exhibited suicidal tendencies and delusions of conjugal infidelity. Three tumours presenting the characteristics of lipomata were discovered in the following sites: angle of the left scapula, left lumbar region, right side of chest in the nipple line just above the costal margin.

Four months after admission, synovial effusion in both knee-joints appeared without known cause, and seemingly suddenly and painlessly. In six weeks the effusion disappeared rather quickly leaving the knees apparently normal.

Three months after admission the original tumours had increased in size and were rather more prominent, while about a dozen others had appeared on the back, the sides, and abdomen. These were of various sizes and various degrees of definiteness of outline. Some were firm and almost fibroid in consistence; others were soft, and some had edges so ill-defined that they appeared to be merely local accumulations of subcutaneous fat without any capsule. Other tumours developed on various parts of the trunk, the face, neck, arms, forearms, thighs, and legs being exempt. The patient died a year after admission from exhaustion following the occurrence of nineteen epileptiform fits in one day. No autopsy was obtained, but examination of one of the smaller and more defined tumours revealed a lipomatous structure.

T. C. MACKENZIE.

**PERFORATING ULCER IN GENERAL PARALYSIS.** (Le mal (186) perforant dans la paralysie générale.) MARIE (of Villejuif) and PELLETIER, *Rev. de Psych.*, Nov. 1905.

SIMILAR conclusions to those of Vigouroux in the preceding article are arrived at; mechanical causes are an inadequate explanation

of the disorder ; it is directly associated with a vasomotor disturbance of nervous origin. In some cases the existence of the ulcer seemed to have a beneficent influence on the psychosis.

C. MACFIE CAMPBELL.

**BED-SORES IN GENERAL PARALYSIS.** (*Les escarres dans la* (187) *paralysie générale.*) A. VIGOUROUX (of Vaucluse), *Rev. de Psych.*, Oct. 1905.

THE author insists on the fact that all bed-sores have not the same origin, and refers to certain clinical observations to show that bed-sores may be directly related to a nervous lesion—cerebral medullary, or neuritic. In general paralysis, bed-sores may develop acutely after an epileptiform or apoplectic attack without pressure or improper care ; in other cases myelitic foci have been discovered.

C. MACFIE CAMPBELL.

**A PSYCHOLOGICAL CONCEPTION OF NERVOUSNESS.** (Con- (188) *ception psychologique au nervosisme.*) H. ZBINDEN, *Arch. de Psych.*, Jan. 1906, p. 185.

THE nervous state includes such conditions as nervousness, hysteria, neurasthenia. It is characterised by primary symptoms, of which fatiguability, anxiety, emotionalism, irritability, and above all, an exaggerated auto-suggestibility, are the chief ; and by secondary symptoms, such as insomnia, palpitation, asthma, dyspepsia.

The causes of the nervous state are discussed under three heads: hereditary, predisposing (education), and determining (fatigue, worry, disappointment, etc).

The consequences of the nervous state on family, social, and political life are discussed at some length, and shown to be multi-form and far reaching, so much so, indeed, that it must seem an almost superhuman task to deal with them. Nevertheless the author does not think the task an impossible one.

The remedy for the evil the author finds in education. At bottom the nervous state is a psychic disorder. Men become "nervous" because they do not think rightly. As our acts are the outcome of our thoughts, it follows that a neurotic does not know how to live. To preserve a nervous equilibrium, one must learn how to think and how to live. If such an education were given with the necessary competence from early childhood, the nervous state would disappear.

In the treatment of disorders arising from the nervous state,

the first point is to gain the confidence of the patient. One must then unravel the disordered train of thought upon which the symptoms depend. Having thus thoroughly mastered the case, one proceeds to explain to the patient how his sufferings have arisen, and when one succeeds in directing his thoughts into healthy channels, and in encouraging him to expect to be cured, the secondary symptoms will disappear one after the other. The main point is, cure the primary symptoms and the secondary will depart of themselves.

The whole paper extends to over sixty pages, and contains many valuable suggestions, but scarcely lends itself to satisfactory summarising.

W. B. DRUMMOND.

**SPASMODIC LAUGHTER AND WEEPING.** (*Le rire et le pleurer* (189) *spasmodiques.*) M. A. DEROUBAIX, *Journal de Neurologie*, March 5, 1906, p. 81.

MOST modern writers agree with Bechterew that the reflex emotional centre is situated in the optic thalamus, but opinions vary as to the connections of this centre with the cerebral cortex and with the bulbar nuclei, and as to the course of the fibres which establish these connections. The writer discusses the views advanced by various authorities, and records two cases observed by himself, with an account of the post-mortem findings in one of them. The first case was that of a coal-miner, aged 67, whose chief symptoms were—difficulty in walking, swallowing, and speaking; slight mental deterioration; loss of control of the sphincters; and frequent attacks of causeless weeping. Examination showed—hesitating, shuffling gait; trembling of hands and tongue; pupils unequal and sluggish; no nystagmus; exaggerated tendon reflexes; absence of Babinski's sign, ankle clonus, and Rombergism; and nothing of note in connection with sensation or the organs of special sense. The outbreaks of crying were quite involuntary, and unaccompanied by any feeling of sadness. Any attempt at a smile was cut short almost before being formed, and its place taken by weeping. The second patient was a miner, aged 38, with a history of apoplexy followed by left hemiplegia two years before, and some recent epileptic seizures. He had paresis of the left side of the face and left arm and leg, with exaggerated tendon reflexes, but there was no ankle or patellar clonus, and no Rombergism. He had no control of urine or fæces. His mental condition was good. He was subject to attacks of weeping exactly similar to those of the first patient. The chief points determined post-mortem were: Slight atheroma of the basal arteries; a small cyst lying between the diverging optic tracts, which had destroyed



the corpora albicantia and softened the pituitary body; and extensive softening of the right side of the brain, the lenticular nucleus, the anterior limb of the internal capsule, and all the white matter of the frontal lobe and central region being destroyed. The author's main deductions are: That the cortical centres for emotional expression are in the frontal and central regions; that the fibres connecting these centres with the optic thalamus run, not in the pyramidal tract, but in front of the genu of the internal capsule, and probably through the corpus striatum; and that a lesion destroying this connection removes the regulating and inhibitory influence of the cortical centres on the lower emotional centre in the optic thalamus, which, being intact, gives rise to involuntary emotional manifestations, *i.e.* to laughing and crying.

HENRY J. DUNBAR.

**ON THE ANATOMICAL AND HISTOLOGICAL BASIS OF SO-CALLED CORTICAL BLINDNESS; ON THE LOCALISATION OF THE CORTICAL VISUAL AREA AND OF THE MACULA LUTEA, AND ON THE PROJECTION OF THE RETINA ON THE CORTEX OF THE OCCIPITAL LOBE.** (Ueber die anatomisch-histologische Grundlage der sog. Rindenblindheit und über die Lokalisation der corticalen Seesphäre der Macula lutea und die Projection der Retina auf die Rinde des Occipitallappens.) EUGEN WEHRLI (Frauenfeld), *v. Graefes Archiv.*, lxii., 2.

IN this elaborate and interesting paper the conclusions reached by Henschen and others regarding the location of the visual areas in the occipital lobe are subjected to searching criticism. Dr Wehrli first describes very carefully the brain of a patient who became suddenly blind after a convulsive seizure with loss of consciousness. In the right half of each field there was a slight return of light perception, but not sufficient vision to make the charting of a field possible. Death occurred about three months after the onset of blindness. At the autopsy there were found softenings in both occipital lobes involving the cuneus, gyrus lingualis, gyrus occipito-temporalis, and præcuneus. Both occipital arteries were occluded by old thrombi. Dr Wehrli specially emphasises the fact that at the post-mortem the impression made was that the lesions were purely cortical. The brain was hardened in formalin and Müller's fluid and cut on the microtome in a series of vertical sections. A very minute description of the naked-eye and microscopical appearances is given. The case might be quoted as a classical example of occlusion of the occipital arteries with con-

secutive necrosis of the cortex in the area supplied by these vessels. In regard to cause, localisation, and extent of cortical softening, this case is therefore exactly comparable with those recorded by Henschen and others. Owing to the comparatively short period between the onset of blindness and the death of the patient, Dr Wehrli claims that in this case there was no time for the development of secondary degenerations, and therefore the extent of the original lesion could be definitely determined.

A detailed and thorough examination of the brain revealed that the supposed purely cortical lesions were not in reality restricted to the cortex, but that on both sides there was an undoubted primary lesion of the optic radiations.

In a series of drawings Dr Wehrli shows that, owing to the depth to which the calcarine fissure penetrates, a lesion on its floor may very readily produce extensive destruction of the fibres of the optic radiations.

Cases of supposed cortical lesions causing hemianopsia, as described by Vialet, Förster-Sachs, Laqueur, and others, are passed in review, and in all of them Dr Wehrli finds evidence of the co-existence of lesions of the white matter and optic radiations. Not a single case, he maintains, can rightly be used to furnish proof on questions regarding the exact cortical localisation of the visual area. He considers, in fact, the cortical lesion relatively unimportant in the production of hemianopsia when compared with the deeper lesions which affect the optic radiations.

In corroboration of this view he quotes nine published cases of extensive lesions of the mesial surface of the occipital lobe in which no hemianopsia was present. In case 7 (Gowers, *Lancet*, 1879), a malignant tumour involved the region of the entire cuneus, upper and middle occipital convolutions, the region of the calcarine fissure and præcuneus, and yet there was no hemianopsia. Apparently, however, Gowers did not see the patient for some months before death, so that the case loses much of its value as negative evidence.

Commenting on these negative cases, Dr Wehrli summarises somewhat as follows:—

1. Whether a softening of the occipital lobe shall cause a hemianopsia or not, depends rather on the number of intact fibres connecting the cortex with the primary optic centres than in the position of the softening in the cortex.

2. The optic fibres, relatively few in number, and mixed with commissural and other fibres, are distributed in fan-like fashion to the occipital cortex. Few come to any one convolution, and their destruction, in the case of small defects, is not sufficient to cause a scotoma.

3. The function of the destroyed projection fibres of the cornea radiata in cases of extensive softening, and in cases where there is

destruction of part of the optic radiations, is taken over by the adjoining fibres and cortical areas, the connection of which with the primary optic centres has remained intact. Countless experiments and a large number of clinical facts support this view.

4. The existence of any sharp projection of the retina on the cerebral cortex (reproduction of the retina on the cortex) is most improbable.

Dr Wehrli is not inclined to lay stress on peculiarities of histological structure as indicative of the visual function being wholly or chiefly limited to any one small cortical area. He instances v. Monakow's experiments on animals, as pointing to a wide area of distribution for the visual fibres.

With regard to the question of the representation of central (macular) vision in the cortex, Dr Wehrli naturally opposes the view of those who hold that the macula is represented in the cortex by a very small area on the floor of the calcarine fissure. He points out, apparently with justice, that direct vision of an object is a function so complex, and involving the co-operation of so many other cortical areas, *e.g.* in forming judgments as to size, taste, smell, uses, and general properties of the object looked at, that its adequate representation in the cortex would require an area larger than that needed to represent the peripheral parts of the retina. He would support v. Monakow's view that fibres from the macula spread themselves over the whole visual area.

On the question of whether different segments of the retina are represented by definite and circumscribed cortical areas, Dr Wehrli holds the view that attempts to map out retinal areas in the cortex are quite fanciful.

It is a well-known fact that in cases of double cortical hemianopsia some restoration of vision almost always takes place, and the portion of the field restored is usually central. This is interpreted by Dr Wehrli to mean that any intact neurons are, as it were, pressed into service to carry on the function of the macula. The failure of restoration of vision in Wehrli's own patient is ascribed not to special severity of the lesion, but rather to the very bad state of the patient's circulation, and the degenerative changes in the cerebral vessels.

In his concluding sentences, Dr Wehrli claims to have shown that purely cortical lesions causing hemianopsia have, so far, never been observed. Conclusions regarding the exact localisation of the cortical visual area and the island-like representation of the macula in the cortex, based on the supposed occurrence of such lesions, do not therefore rest on any firm foundation of fact.

The article is amplified by a series of plates illustrating the lesions in the present case, and in a large number of important cases published by various observers.

J. V. PATERSON.

**ISOLATED PALSY FOR LATERAL MOVEMENT OF AN INTERNAL RECTUS MUSCLE.** (Isolierte Lähmung eines Muskels rectus internus als Seitenwender.) OSKAR FISCHER, *Prag. med. Wochenschrift*, xxx., No. 49, 1905.

DR FISCHER quotes briefly two cases recorded by Bielschowsky where the internal rectus of one side failed to act in lateral movement while convergence was unaffected. He then cites a case which came under his own observation. Patient, æt. 42, a sufferer for some years from symptoms pointing to multiple sclerosis, was seized with a sudden attack of giddiness; objects appeared doubled when he looked to the left, and he had at the same time the feeling of a sort of jerk or wrench in the left eye. On examination, a week later, movements upwards, downwards, or to the right were normally executed, but when an attempt was made to look to the left the right eye failed to move inwards more than  $25^{\circ}$ , while the left eye, after a moment's pause, suddenly took up a position of extreme outward rotation. The patient was at the same time conscious of an unpleasant jerk in the left eye and a crossed diplopia appeared. With the right eye covered, the left eye moved readily and smoothly to the left, the right eye behind the occlusion screen failing to move inwards beyond an angle of  $25^{\circ}$ . No feeling of jerk in moving the eye was felt as long as the right eye was covered. If, on the other hand, the left eye was covered, the right eye failed as before to move inwards beyond  $25^{\circ}$ , while the left eye behind the screen made a sudden movement outwards into the position of extreme abduction and the patient had the same sensation of a jerk. Convergence movements were completely intact, and also when an object near the eyes was moved a little to the left the right eye was able to assume a position of extreme inward rotation.

In less than a month the internus paresis had completely disappeared. The lesion must have been so placed as to interrupt the stimulus passing to the internal rectus while leaving intact the path to the corresponding external rectus. At the same time the path for the passage of convergence stimuli to the internal rectus must have been intact. The position of such a lesion is indicated by a diagram.

J. V. PATERSON.

**A STUDY OF THE CONTRACTURES IN ORGANIC NERVOUS DISEASES, AND THEIR TREATMENT.** T. H. WEISENBURG, *Univ. of Pennsylvania Med. Bull.*, 1905.

THIS paper is based upon the study of 500 cases of nervous disease. The author divides contractures into two great classes, passive and

active. By passive contracture he indicates a condition due to disease of the part, such as joint disturbance, inflammation round a joint, paralysis of antagonistic muscles, as, for instance, in multiple neuritis and infantile paralysis, etc. Passive movement of such contracted limbs is either impossible or very limited in extent, and the contractures are not influenced by foreign conditions, such as heat, and do not disappear during sleep or narcosis. Active contractures are always due to disease of the central nervous system. Passive movement is always possible, and during sleep or narcosis the contractures may disappear, provided no structural changes are present. Passive contracture may become super-added to active contracture when such structural changes supervene.

In writing of contractures in cerebral diseases he states that early contracture is only possible if the motor tracts are not completely cut off, and that its occurrence warrants one in giving a better prognosis. Variations from the usual type of hemiplegia contracture are not infrequent, and one of the most common is extension of the fingers instead of flexion. This is seen especially in infantile hemiplegia. In bilateral contractures of cerebral origin both thighs are invariably flexed. He holds that in adults, at any rate, such contractures are never due to a unilateral lesion, as has been mentioned by some members of the French school. The various forms of contracture met with in Little's disease and diplegia, and also in diseases of the spinal cord, peripheral nerves and muscles, are all analysed and described.

The author mentions some of the various hypotheses which have been advanced as to the causation of contractures in cerebral disease, but he does not commit himself to any special theory. The return of function in the paralysed limbs after destruction of the pyramidal fibres is, in the author's opinion, best explained by the influence of the extra-pyramidal tracts. This view was brought forward by Rothmann as the result of his experimental research, and has been supported by the clinical and pathological observations of Von Monakow, H. v. Alban, and M. Lufeld. Reference is also made to the condition "hemitonia apoplectica" of Bechterew, or "hemihypertonia post-apoplectica" as named by Spiller, which is due to irritative lesion of the extra-pyramidal tracts.

The last portion of the paper deals with the treatment of contractures, and is eminently practical and full of sound advice.

T. GRAINGER STEWART.

**SENSORY APHASIA, WITH RIGHT HOMONYMOUS LATERAL (193) HEMIANOPIA.** (*Aphasie Sensorielle avec hemianopsie latérale homonyme droite.*) DEBRAY, *Journ. de Neur.*, Jan. 20, 1906, p. 21.

AN interesting discussion is added to the description of a case of sensory aphasia, coupled with right homonymous lateral hemianopia. The patient was a man fifty-seven years of age, who one day, when riding a bicycle, was observed suddenly to lose control of it, and was found, when interrogated by his friends, to be talking nonsense. There was no loss of consciousness. When examined later he was seen to present a right homonymous hemianopia. He understood what was said to him, and could answer simple questions, could read print and writing, and was able to write himself. He could not, however, spontaneously express any idea either in words or in writing, repeating indefinitely a short phrase without perceiving he was doing so. His speech was frequently paraphasic, and indeed jargonaphasic.

The lesion was probably located in the subcortical region of the occipital lobe, destroying the fibres joining the cortex in the neighbourhood of the angular gyrus to the motor paths and to the motor vocal centres, as well as those in the area round the calcarine fissure.

There was not, however, any deviation of the head and eyes, yet there ought to have been, if Bard's view of the phenomenon as a sensory reflex is correct, the deviation being due to loss of visual sensations in the altered portion of the cortex. As the cortical cells on the lips of the calcarine fissure were probably unaffected in this case, the fibres uniting them to the centre for conjugate movements of the head and eyes may possibly have been untouched.

As in many cases of hemianopia, the patient presented the peculiarity of being able to distinguish differences of light in the field in which he could no longer discern objects or colours. According to Bard, the visual sensations of light, form, and colour may be compared to the cutaneous sensations of touch, temperature, and pain, and these modalities of vision depend on the activity of the cortical cells of the occipital pole. Morat believes in the existence of homolateral retinal fibres concerned with the "crude" sensations of light and darkness. According to the author, all the cells of the occipital lobe are capable of appreciating the stimulus of luminous waves, whereas those of the occipital pole are in addition able to distinguish the form and the colour of objects, because they are more directly in continuity with the retinal cones constituting the fovea.

Pure hemiachromatopsia and hemiamblyopia are rather the

result of a functional alteration of the occipital cells than of their destruction. Corroboration of the author's view would probably be found in a unilateral complete lesion of the bandelettes. It has been shown by Déjérine and Gialet that hemianopia resulting from such a lesion is much more complete than that caused by disease in the occipital pole.

S. A. K. WILSON.

**PHILIPOVICZ'S SIGN IN ENTERIC FEVER.** (*Il sintoma palmo-plantare di Philipovicz nella febbre tifoidea.*) G. MINCIOTTI, *Gazz. degli Osped.*, March 25, 1906, p. 376.

PHILIPOVICZ, in 1893, described as pathognomonic of enteric fever an orange-yellow coloration of the palms and soles which occurred at an early stage of the disease. Subsequent observers showed that the condition, though not peculiar to enteric fever, was more frequently found in that disease than in any other. The phenomenon was attributed to a trophic alteration of the epidermis, due to elimination through the skin of the typhoidal toxins. Though sensory disturbance in the limbs in the course of enteric fever is not infrequent, there has hitherto been no record of local pain being associated with Philipovicz's sign.

Minciotti's patient was a woman, aged 26, who, at the end of the first week of a hitherto uneventful attack of enteric fever, complained of severe pain and tenderness in the metatarso-phalangeal region of the left sole. There was no local evidence of inflammation, but the part had assumed an intense orange-yellow colour. The corresponding region of the right foot had a similar, though less intense, coloration. Spontaneously it was completely painless, but slight pressure provoked a feeling of discomfort. There was no sensory disturbance in the hands, but the thenar and hypothenar eminences and thumb of the left palm had a yellowish coloration. The right palm was not affected. The pain in the left sole continued to be troublesome for another week, proving rebellious to all treatment. Spontaneous pain subsequently developed in the corresponding coloured zone of the right foot, but was of moderate intensity and short duration. The coloration gradually diminished, so that when convalescence was attained no traces were found in the left hand or right foot. In the left foot the coloration persisted for a month longer, gradually fading away *pari passu* with the sense of discomfort which had succeeded the more severe symptoms. Minciotti attributes the pain to a disturbance of nutrition in the terminations of the sensory nerves in the regions affected, the disturbance being due to weakening of the cardiac action characteristic of the typhoid infection.

J. D. ROLLESTON.

**PSYCHIATRY.**

**AN ANALYSIS OF INSIGHT IN MENTAL DISEASES.** (Zur (195) *Analyse des Krankheitsbewusstseins bei Psychosen.*) E. ARNDT (of Greifswald), *Centralbl. f. Nervenl. u. Psych.*, Oct. 1905, p. 773.

INSIGHT into any disorder is only possible under certain conditions: there must be unusual feelings and sensations due to the disordered working of the organ, these feelings must be correctly appreciated and not distorted or fantastically explained, and there must be a certain familiarity with the nature of disease.

Sane people do not always have good insight into their physical condition; the absence of insight of the insane is not always pathological.

Arndt discusses the influence on the presence of insight of the various psychopathological symptoms, dividing these into intellectual, emotional, and volitional.

Frequently the very nature of the intellectual disorder, *e.g.* the false interpretation and elaboration of changes dependent on subjective conditions, excludes the possibility of insight. In the life of feeling, pain is more importunate in its demand for explanation than pleasure; pain is something external and foreign to the individual, and insight is more common in conditions dominated by distressing emotions than in those where the disorder takes the form of an exhilaration: the latter seems to the patient to belong to his personality. Volitional disorders not infrequently occur where the general judgment is fair, and are frequently recognised as pathological.

The question of insight is apt to be exaggerated from the point of view of diagnosis, prognosis, and treatment; its value depends entirely upon its relation to the various psychopathological elements in the psychosis.

C. MACFIE CAMPBELL.

**MENTAL CONFUSION.** (*La confusion mentale.*) E. RÉGIS (of (196) Bordeaux), *Ann. méd. psych.*, Sept.-Oct. 1905.

RÉGIS endeavours in this article to trace the clinical picture of mental confusion, a well-defined group according to the French school, but one which contains, according to the school of Kraepelin, cases belonging to quite different groups, some cases coming under the infective and exhaustion psychoses, others under dementia præcox. The work of Chaslin has had great influence on the French school, and Régis begins with his definition; his own description of this condition is that it is "a generalised



psychosis, characterised by a toxic dulness and torpor of higher mental activity, sometimes to the extent of its suspension, and accompanied or not by an automatic dream-like delirium, with adequate reaction of the general activity and of the various functions of the organism." It is essentially a toxic psychosis; headache is one of the most prominent physical symptoms; mental torpor and a tendency to dream-like delirium are present, and either may dominate the picture. The course is variable; it is the most curable of all the psychoses, but may be fatal; if it becomes chronic, the typical deterioration is that of dementia præcox. While the symptomatological description by Régis of these toxic cases is adequate, he passes by the difficult questions of their relation to similar cases with no known intoxication or infection, and of the relation of the chronic cases to the whole dementia præcox group.

C. MACFIE CAMPBELL.

**MIXED APRAXIA.** (Ueber einen weiteren Symptomenkomplex im (197) Rahmen der Dementia senilis bedingt durch umschriebene stärkere Hirnatrophie [gemischte Apraxie].) PICK, *Monatsschr. f. Psych. u. Neur.*, Bd. xix., H. 2.

PROF. PICK describes a case of senile dementia with post-mortem results. The case presented clinically "mixed" (ideo-motor + purely motor) apraxia and amnesic aphasia with general psychic enfeeblement. The author comments on the Temporal-lobe-symptom-complex (*Schläfelappenkomplex* of Stransky), and shows that in this case there was no diffuse or general atrophy, the atrophy being most pronounced in the L. Inf. Par. Lobule and in the Frontal convolutions of both sides, and to a less degree in the Temp. Lobes of both sides, most marked in the 2nd and 3rd L. Temp. The central convolutions of both sides, both Precunei and Cunei, were intact, and only the L. Lat. Ventricle and the 3rd Ventricle dilated. The author considers that in all probability the frontal atrophy did not contribute to the apraxia, but occasioned merely the general mental weakness, and discusses the relation of the L. Parietal and Temporal atrophy to the apraxia, and the coincidence of the atrophic areas with Flechsig's associative centres.

R. CUNYNGHAM BROWN.

**SLIGHTLY ABNORMAL CHILDREN.** (Leicht abnorme Kinder.) (198) E. THOMA (of Illenan), *Allg. Ztschr. f. Psych.*, Bd. 62, H. 4.

THOMA discusses the neuroses and mild psychotic disturbances which occur in childhood. He first describes the cerebral neuras-

thenia of children, a condition usually due to over-pressure in school work. The typical symptoms are a general languor and want of interest, a diminution in mental efficiency, emotional anomalies; vasomotor disorders are prominent, patient has unpleasant head-feelings. The condition occurs in children with poor heredity, and runs a favourable, although tedious, course of several months. The treatment is to regulate the child's activities and allow plenty rest. As evidences of a psychopathic disposition, compulsive ideas and phobias are frequently met with; a pedantic scrupulousness in children is very suspicious. It is frequently advisable to remove such children from their home environment. As for the tics which are so frequent in children, treatment by suitable gymnastics is recommended; punishment is always injurious. Among psychotic symptoms are to be regarded the exaggerated day-dreaming of some children, their wilful romancing, and some truant episodes. The hysteria of children is essentially the same as that of the adult, but, owing to its frequently monosymptomatic appearance, the diagnosis is frequently difficult; from this point of view various coughs, pains, twitchings, stuttering, etc., are important. The conditions occurring in childhood are extremely varied, but almost all point to an inherited psychopathic constitution. The function of the school physician in picking out such children, and the necessity of suitable schools and institutions for such children are finally insisted upon.

C. MACFIE CAMPBELL.

#### LATE EPILEPSY IN THE COURSE OF CHRONIC PSYCHOSES.

(199) (*Die Spätepilepsie im Verlaufe chronischer Psychosen.*)

P. NÄCKE (of Hubertusburg), *Allg. Ztschr. f. Psych.*, Bd. 62, H. 5, 6.

THE author excludes cases where epileptic attacks developed on a syphilitic or an alcoholic basis, or where there was a history of attacks in early life. Epileptiform attacks are not very uncommon in the acute stage of dementia præcox, but are very rare in the case of chronic psychoses, except on the bases mentioned above. The author reports thirteen cases in some detail. He considers that these attacks are genuine epileptic attacks; they rarely occur in rapid succession, and were rarely replaced by equivalents in the form of attacks of dizziness. They came on in the cases reported from six to fifteen years after admission to the Hospital; the interval between the attacks was sometimes several years. An aura was rarely present; post-epileptic symptoms were the rule.

Näcke considers the late epilepsy to be one symptom of the

psychosis, which has so prepared the soil that the exciting cause, whatever that be, is able to discharge the epileptic attack.

C. MACFIE CAMPBELL.

**THE DEMENTIAS: PATHOLOGICAL ANATOMY AND PATHO-**  
(200) **GENESIS.** (*Les démences: Anatomie pathologique et*  
*pathogénie.*) KLIPPEL and LHERMITTE (of Paris), *Rev. de Psych.*,  
Dec. 1905.

AFTER some general considerations the authors give an outline of the cortical changes in the dementias, which they divide into three groups. In the first group, including the various forms of dementia præcox, the vessels are not implicated; there are some changes in the glia, and the cortical cells, especially in certain regions, show a definite form of degeneration. In the second group, the dementias of the adult, which include a variety of conditions, both the mesodermal and the ectodermal elements are affected as a rule, the cause usually being some form of intoxication; in certain dementias of more endogenous origin only the ectodermal elements may show changes. In the senile one meets two forms of dementia, which have both clinical and pathological characteristics; in the one the degeneration is limited to the ectodermal elements, in the other the vessels also show a definite reaction.

A number of observations are given, with pathological reports.

C. MACFIE CAMPBELL.

## TREATMENT.

**TREATMENT OF THE TICS AND CHOREAS OF CHILDHOOD.**  
(201) (*Traitement des chorées et des tics de l'enfance.*) BRUEL,  
*Thèse de Paris*, 1906. Paris: Steinheil.

THERE is little, if anything, novel in the discussion of the therapeutic measures in vogue for the choreas and tics of childhood, as presented by the author. Systematic scanning of the literature has enabled him to specify very numerous procedures, of which personally there is little evidence of experimental knowledge. The methods of which most details are vouchsafed are alimentation and isolation, in association with psycho-motor discipline. In the chapter, or rather paragraphs, on the rheumatic theory of chorea, no reference is made to the work of English observers, and an eclectic position is adopted which scarcely harmonises with recent advances in our knowledge of the subject.

S. A. K. WILSON.

**DIETETIC TREATMENT IN EPILEPSY.** WILLIAM ALDREN  
(202) TURNER, *Practitioner*, April 1906.

IN a short paper the author reviews the results obtained in the treatment of epilepsy by modifying the diet, and brings forward a dietetic formula which has been followed by considerable success in his hands.

Alt concluded (*Zeitschr. f. klin. Med.*, vol. liii.) that a diet without meat was the most satisfactory, but that neither a milk diet alone nor a vegetable diet was as beneficial as their combination.

"Salt starvation" has proved a useful adjuvant to bromide medication in some cases, while in others little benefit has resulted. The author has recorded in a previous number of this journal (*Rev. of Neurol. and Psychiat.*, 1904, p. 793) his experience of the salt starvation diet of Toulouse and Richet as modified by Balint. He has recently tested the efficacy of a *purin-free* diet. Tea, cocoa, coffee, and all kinds of fish, fowl, and meat (including sweetbread) are to be avoided. Tripe, neck of pork, and cod-fish may be given, since they contain only relatively small quantities of purin bodies. Peameal, malted lentils, potatoes, and onions, although they contain small quantities of purin bodies, may be given. Milk, eggs, butter, cheese, rice, macaroni, tapioca, white bread, cabbage, lettuce, cauliflower, sugar, and fruit are free from purin bodies, and are, therefore, suitable.

Details of five cases are narrated in which distinct improvement was noticeable when the patients were placed on a purin-free diet in addition to a dose of 20 or 30 grains of sodium bromide at bedtime. In every instance there had been previously a more or less prolonged course of bromides, with little or no improvement.

The author concludes from these results that "the elimination of the purin element from the dietary of epileptics is of great therapeutic assistance in the treatment of the disease."

EDWIN BRAMWELL.

**ON BLEEDING IN EPILEPSY.** (*De la saignée dans le mal épileptique.*) GH. HOUZEL, *Presse Méd.*, Jan. 31, 1906, p. 67.

THE author has had two cases of status epilepticus which he successfully treated by bleeding. In the first case, breathing had practically ceased and the respiratory passages were obstructed by a fine froth. The pulse was imperceptible, there was cyanosis, and the extremities were cold. Bleeding was practised rather as a forlorn hope. In a quarter of an hour recovery occurred. The second case had been in an epileptic condition for four hours

when he was bled and immediately recovered. The first case was a little girl in whom epilepsy would not have been expected; the second, a boy with marked stigmata of idiopathic epilepsy. In both cases bleeding stopped the fits, lowered the arterial pressure, slowed the pulse, and was followed by freedom from symptoms for several days.

ALEXANDER GOODALL.

**PREPARATION OF A SERUM FOR THE TREATMENT OF**  
(204) **EXOPHTHALMIC GOITRE.** S. P. BEEBE, *Journ. Amer. Med. Assoc.*, 1906, Vol. xlvii, p. 484.

**THE TREATMENT OF EXOPHTHALMIC GOITRE BY A SPECIFIC**  
(205) **SERUM.** JOHN ROGERS, *Ibid.*, p. 487.

THE sera used in the treatment of ten cases of exophthalmic goitre were obtained after injecting rabbits with the nucleo-proteids and thyreoglobulin isolated from the thyroid glands of two cases of exophthalmic goitre. The method of isolating the nucleo-proteids was described in a former paper by Beebe (*Journ. of Exper. Med.*, Nov. 1905). The globulin was precipitated by half-saturation with ammonium sulphate, and after filtration and washing the excess of salt was removed by dialysis. The nucleo-proteids were included in the mixture injected into the rabbits, because it was desirable to produce some cytolytic effect on the thyroid gland, and the globulin because it was hoped thereby to develop an antitoxine to counteract the toxic symptoms of exophthalmic goitre. The rabbits were given injections of the two proteids intraperitoneally at five-day intervals. On the eighth day after the last injection the animals were bled from the carotid artery.

Ten cases of exophthalmic goitre were treated by subcutaneous administration of the sera, "with a result of three apparently perfect cures, three rescued from a critical condition and now approaching a cure, and the others more or less improved."

W. T. RITCHIE.

**RECENT SURGICAL METHODS IN THE TREATMENT OF**  
(206) **CERTAIN FORMS OF PARALYSIS.** A. H. TUBBY, *Brit. Med. Journ.*, March 3, 1906.

THE forms of paralysis chiefly dealt with are those arising from anterior poliomyelitis, spastic paralysis, and traumatic nerve lesions. The methods discussed are tendon and muscle transplantation, arthrodesis, and nerve anastomosis; the results of tenotomy and of lengthening and shortening of tendons are regarded as too well known to require description.

In *tendon and muscle transplantation* the reinforcing tendon may be completely divided, and its central end inserted into the tendon of the paralysed muscle; or the reinforcing tendon may be split, and only a strip transplanted. In the latter case it is difficult to obtain functional independence of the strip, while in both methods the two tendons must be comparatively near one another. Many of the results are ultimately disappointing, owing to stretching of the paralysed tendon. A great improvement was effected by stitching the reinforcing tendon directly to the periosteum, which avoided this stretching, and gave an attachment experimentally proved to be five or six times stronger. But in many cases the reinforcing tendon or muscle is too short for this purpose, hence the value of Lange's method of prolonging the tendon by means of silk threads which bridge the gap between the end of the tendon and the selected point of periosteal insertion. Four to eight strands of silk are employed, and subsequent sections show that the silk becomes enveloped and infiltrated by new tissue, which thickens with use. In this way intervals of from eight to ten inches have been successfully bridged, and the most favourable site of insertion can be chosen; thus, in paralysis of the quadriceps extensor, the hamstrings can be transplanted by means of these silken tendons into the tubercle of the tibia instead of into the patella. Successful or encouraging results have been obtained in the various forms of club-foot due to infantile paralysis; in paralysis of the quadriceps extensor, by transplantation of the hamstrings, sartorius, or ilio-tibial band; in dropped wrist, by conversion of the carpal flexors into carpal extensors; in paralysis of the biceps, by use of a strip of triceps; and in scapula alata, by grafting part of the pectoralis major into the serratus magnus. In spastic paraplegia and cerebral diplegia the technical difficulties are greater and the results less encouraging. Great improvement has often been secured, but simple tenotomies are in many cases similarly successful. For tendon transplantation a preliminary correction of secondary deformities is essential, and the reinforcing and the reinforced muscles should, if possible, be synergic. Cases of extensive paralysis should not be attempted, and a slender muscle like the peroneus cannot be expected to perform the function of paralysed calf muscles. Reinforcement, by removing the constant stretching, often allows an apparently paralysed muscle to recover.

*Arthrodesis* is indicated where a joint is hopelessly flail-like; it merely aims at restoration of stability, but may be accompanied by transplantations.

In *nerve anastomosis and transplantation* an interesting field is opened up. Experimentally it has been proved that the central end of an efferent nerve fibre can make functional union with the

peripheral end of any other similar efferent fibre. The nerves to flexor and extensor groups of muscles may thus be interchanged and will take on their new function, the cortical areas for flexion and extension also becoming interchanged. Where one central nerve trunk is made to supply two distal ones, an actual division of nerve fibres takes place. Regeneration of the muscles supplied may set in even eight years after atrophy. Very successful cases of facio-hypoglossal anastomosis for facial paralysis are recorded, the hypoglossal being preferred to the spinal accessory because its action is more associated with that of the facial, and less after-education is therefore required. The obturator nerve has been called upon to take up the work of the anterior crural, and the nerves to soleus and gastrocnemius have been transferred from a paralysed internal to a healthy external popliteal nerve, with fair success. In some cases considerable power returned, without return of faradic excitability.

In both tendon and nerve transplantations prolonged subsequent care and education are required, but the results are sufficiently encouraging to justify careful trial.

W. J. STUART.

**TREATMENT OF SELECTED CASES OF CEREBRAL, SPINAL, (207) AND PERIPHERAL NERVE PALSIES AND ATHETOSIS BY NERVE TRANSPLANTATION.** SPILLER, FRAZIER, and VAN KAATHOVEN (of Philadelphia), *Am. Journ. of Med. Sciences*, March 1906.

NERVE transplantation in cases of acute anterior polio-myelitis, to bring diseased peripheral nerve fibres into union with healthy nerve fibres, gives more advantageous results than tendon transplantation. If the operation is successful, the function of the paralysed muscles should be restored. The chief dangers are delayed union, and overgrowth of connective tissue in the nerve at the site of operation. The most favourable cases are those in which the paralysis is confined to a small group of muscles. Spiller's case, reported in the *Journal of Nervous and Mental Disease*, June 1903, showed some return of power in two months, and distinct improvement in two years. Hackenbruch, in June 1903, reported two cases in which partial success followed the insertion of a third of the tibial into the paralysed peroneal nerve; he reported a third case in which marked improvement followed the insertion of one-half of the posterior tibial nerve into the peroneal nerve after one and a half years.

In cases of athetosis the flexors are, as a rule, much more powerful than the extensors; for this reason Spiller thought that

an attempt to establish an equilibrium by switching off some of the flexor power into the extensors by nerve transplantation was justifiable. Frazier operated upon a young man who had shown violent athetoid movements of the upper limbs from infancy; the operation consisted in a lateral anastomosis in left arm of the divided median and ulnar nerves with the musculo-spiral; two and a half months later there was a distinct return of power in the wrist, in the flexors of the fingers, and at the elbow, but the movements were far from normal. Seven and a half months later the improvement was most encouraging. With the partial paralysis due to operation on the nerves, athetosis had seemed to disappear. Sensation for touch and pain were present over the front and back of the forearm, but less acute than in the right forearm; sensation for touch and pain in the palmar surface of the hand was lost. A second operation on the same arm consisted in an end to end anastomosis between the cut circumflex and musculo-cutaneous nerves (central end of one to the distal end of the other and vice versa). This was successful, inasmuch as it seemed to ease the violent contraction of the shoulder muscles.

Frazier reviews the physiological law upon which is based the operative treatment of palsies by nerve transplantation. He discusses briefly the best method of effecting anastomosis, and describes the operative technique. Excellent photographs and drawings are introduced to illustrate the paper.

C. H. HOLMES.

---

## Reviews

**HISTOLOGISCHE UND HISTOPATHOLOGISCHE ARBEITEN  
ÜBER DIE GROSSHIRNRINDE MIT BESONDERER BE-  
RÜCKSICHTIGUNG DER PATHOLOGISCHEN ANATOMIE  
DER GEISTESKRANKHEITEN.** Edited by FRANZ NISSEL,  
Professor of Psychiatry in Heidelberg. Vol. i. With 14  
plates and 23 illustrations in the text. Jena : Gustav Fischer.  
1904. Price 40 M.

THIS work is the first of two volumes in which Nissl proposes to publish the results of various cortical studies carried on by him and his pupils in the laboratory at Heidelberg, and by Alzheimer partly at Heidelberg, but chiefly at Frankfort. The first volume is devoted to General Paralysis, and contains two articles embody-



ing the results of Alzheimer and of Nissl, who worked at the same subject independently and on separate material.

Alzheimer writes on "Histological Studies towards the Differential Diagnosis of General Paralysis" (pp. 18-314), and his work is accompanied by fourteen plates and twenty-three illustrations in the text.

Nissl follows with an article on "The Histopathology of the Cortical Changes in General Paralysis" (pp. 315-494); he refers to Alzheimer's drawings, but reserves his own for the second volume.

Alzheimer begins by defining his general aim in carrying on his histopathological researches; he hopes by establishing the histological characteristics of the various disease-processes to enable the clinician to define more clearly the limits of the various disease-groups. General paralysis, with its marked tissue changes, offers a good starting-point.

The material used was derived from 320 successive autopsies in the Frankfort asylum; of these, 170 were cases of general paralysis. The largest section of the work is occupied with the description of the macroscopic and microscopic changes in general paralysis, their nature, distribution, and meaning, and the contribution which this knowledge makes to our clinical grouping of the cases. The macroscopic changes are first discussed, and then the histological changes are taken up in detail. The membranes first receive attention; in every case there is present in the pia a cellular infiltrate which is not merely perivascular; new formation of capillaries is rare. In the cortex the vessels always show proliferative changes, and there is an increase of vessels in all but the most acute cases; this much disputed point can be regarded as settled by the researches here published. In every case there is diffusely distributed throughout the cortex a perivascular infiltrate of plasmon cells. In the parenchyma itself a rod-shaped cell is regularly present (*Stäbchenzelle* of Nissl); it is more picturesquely and accurately called a "sausage cell," and is derived from the vessel wall. Granular cells were not found apart from focal destruction, and polymorphonuclear leucocytes always denoted secondary infection.

The changes of the nerve-cells in general paralysis are bewildering in their variety; they are probably not meaningless degenerations, although very difficult of interpretation; in advanced cases there is always a considerable disappearance of nerve-cells.

In that portion of the parenchymatous tissue which our technique stains very imperfectly, the "nervous grey" of Nissl, degenerative processes take place.

The glia always shows proliferative changes, and the various

progressive and regressive changes are illustrated by chromolithographs in which the author's drawings are admirably reproduced (plates viii.-xi.): the staining methods used were mainly those of Weigert, Nissl, and Bevan Lewis.

The next question taken up is that of the topographical distribution of the changes in the nervous system, and in this context he records eight cases presenting focal symptoms without actual foci of softening or hæmorrhage, but with marked severity of the paralytic changes in circumscribed areas. The clinical course of such cases is usually spasmodic, the attacks occurring after long intermissions and leaving the same defect symptoms.

Alzheimer calls attention to the histological changes in the thalamus, where one finds not only primary changes, but occasionally also degeneration secondary to the cortical changes; it is a puzzling fact that the pulvinar is always the region with most marked degeneration, for it has no known relations to the cortical regions most affected.

The changes in the cord are next examined. Histologically one cannot differentiate the paralytic from the tabetic degeneration of the posterior columns of the cord; but in the former the endogenous systems are early attacked in opposition to what occurs in tabes. It is a primary degeneration, and can not be explained as secondary to vascular changes. This is important in discussing the essence of the paralytic process. The distribution of the paralytic disease-process cannot be explained by the inflammatory changes (*i.e.* "inflammatory" in the sense of a cellular exudate); although in the cortex it does not follow any known systemic distribution, it is at least much influenced by the nervous architecture of the cortex.

Histological data do not enable us to demonstrate varieties of general paralysis corresponding to the usual clinical groups; Binswanger's hæmorrhagic and meningo-encephalitic forms have doubtful value.

In the next section (pp. 158-198) the histological differential diagnosis is discussed between general paralysis and various syphilitic conditions, arterio-sclerotic brain atrophy, and the changes in chronic alcoholism and senile insanity. Special attention is paid to the meningo-gummatous variety of brain syphilis and to syphilitic endarteritis of the small cortical vessels (Nissl). In this latter, in contrast with general paralysis, there is much greater proliferation of the cells of the vessel-walls, absence of infiltration of the lymph-sheaths (unless there is focal softening), less destruction of the parenchyma, a different distribution of the morbid process, and a greater general tendency to focal softening.

The last section (pp. 198-292) is the part which is of most

value to the clinician, and can be read with much advantage, even should the earlier detailed pathological discussions be found difficult of digestion by those who have done little laboratory work. Here Alzheimer endeavours to correlate his histological results with the clinical phenomena, and he records a series of cases.

The importance of a careful detailed psychiatric analysis of these cases of difficult diagnosis is brought out; the focal symptoms and associated neurological symptoms are of diagnostic value, but equally essential are the clinical course, the type of dementia, the nature of the affect, the interests, the memory changes, and abnormal mental trend of the patient.

As yet we have, from want of recorded material, no definite clinical picture to correlate with the syphilitic endarteritis of the small brain-vessels (Nissl). Through the microscopic isolation of this pathological process we may hope soon to establish its clinical symptoms. With regard to arterio-sclerotic dementia, the author emphasises its existence as a nosological entity, and refuses to consider it a transition form between alcoholic and senile dementia; it has definite clinical and pathological characteristics, is less a cortical than a subcortical affection, and may be described as an association dementia.

In conclusion, the author mentions certain questions associated with idiocy that await solution, and points to the limits of our present knowledge. No one can deny the claim in the closing sentence that the results of these investigations demonstrate the value of histopathological work for clinical psychiatry.

In the beginning of the second article of the volume, Nissl states the question before him thus: Can we, on the basis of the anatomical examination alone, decide that a case has been one of general paralysis? The answer he gives is that in general paralysis there is a definite histopathological picture.

It is evident that Nissl is treating the same question as Alzheimer, but his work is in many respects complementary to that of the latter. Thus Nissl leaves to Alzheimer the detailed description of the progressive and regressive changes in the vascular elements while he himself takes up the question of the nature and origin of the cells in the perivascular infiltrate and of other mesodermal elements. While he revels in the extremely detailed analysis of the mesodermal and ectodermal elements in the paralytic cortex, he does not confine himself to mere morphology, but discusses in a suggestive and stimulating way the wider biological and pathological questions involved.

He begins with a short sketch of the development of his own views on general paralysis. In 1896 he held that general paralysis is a primary affection of the cortical neurones, and may develop

without any affection of the vessels and with no inflammatory changes. He now holds that there is an inflammatory process present, and agrees with Alzheimer that in every case the vessels are affected and present a perivascular infiltrate. The diagnosis of general paralysis can be definitely excluded if there is not present a diffuse plasma cell infiltrate in the cortex.

Before passing to the plasma cells he discusses the nature of the "rod cells" (*Stäbchenzellen*), to which he first called attention, in the paralytic cortex; these he now holds to be of mesodermal origin. In general paralysis, granule cells are, as a rule, absent, unless there is some hæmorrhage or softening; but these elements are of such importance in the histopathology of the cortex that Nissl devotes eleven pages (pp. 329-340) to their morphology and histogenesis. These are the epithelioid cells of Friedmann, the granule cells of other authors; Nissl calls them mesh-work cells (*Gitterzellen*) on account of their morphological features. They are of mesodermal origin, and are not hæmatogenous; they are the phagocytic cells *par excellence*, and also the elements that fill up gaps in the tissue due to focal destruction.

The presence of the rod cells and Gitter cells and occasional plasma cells in the parenchyma, and other facts, have forced Nissl to give up his old view, that the adventitia of the vessel wall continues to act as a biological limiting membrane between mesodermal and ectodermal elements even in pathological conditions. The hæmatogenous elements, however, are kept back with few exceptions, and the infiltrate remains perivascular and does not invade the ectodermal tissue. The majority of the cells in this infiltrate are cells which Nissl was the first to identify with the plasma cells of Unna. The origin, nature, function, and destiny of these cells have given rise to an enormous literature, of which an exhausting and exhaustive review is given (pp. 347-361). This is followed by an analysis of their morphology (pp. 362-379) and a general discussion of their diagnostic importance. Nissl holds that these cells are of hæmatogenous origin, being modified lymphocytes; his opinion is largely based upon experimental work on the production of tubercle in the animal cortex. He admits that his conception of plasma cells is unusually broad. The important characteristic of a plasma cell is, according to him, that the cell body stains extremely deeply with methylene blue, the dark substance being not sharply defined granules, but rather a crumbly substance, between which clearer spaces are to be seen. The cell body has most morphological importance, the peripheral arrangement of the nuclear chromatin and the eccentric position of the nucleus being non-essential. The practical importance of the demonstration of a plasma cell infiltrate is great, for it enables the physician, although not an expert pathologist, to definitely

place his case within a certain very limited group of psychoses. In fact, it usually establishes the diagnosis, for the other conditions with plasma cells, such as tubercular and carcinomatous processes, acute and sub-acute forms of encephalitis and certain forms of idiocy and epilepsy are not liable to lead to confusion; while a diffuse gummatous meningo-encephalitis presents various histological points of difference.

With regard to the nerve cell changes, Nissl agrees that the variety is disheartening, but he suggests lines of experimental study. He then gives an exceedingly thorough discussion of the glia question, reviewing the views of Weigert and Held, and describing in detail the progressive and regressive changes and the regressive changes in progressively altered cells. He hesitates to accept all of Held's views, *e.g.* his *membrana limitans* and his view of the Golgi net, but he is in sympathy with his view of the glia as a syncytial tissue.

In summing up, he repeats that general paralysis is an inflammatory condition, if the term inflammatory be applied to processes where, along with progressive and regressive changes in the parenchyma, there is implication of the vessels in the sense of a cellular infiltrate; and if one considers the plasma cells of the paralytic exudate to be of hæmatogenous origin. Having discussed the inflammatory aspect of the morbid process, he next turns to the degenerative process which accompanies the former. He regrets that his method of working on little blocks of tissue does not enable him to form a definite opinion on the complete distribution of these changes, and insists on the importance of studying this distribution in large brain sections.

However great the value of the inflammatory changes from the diagnostic point of view, they do not explain the morbid process and the degenerative element. In this context the relation of general paralysis to various forms of brain syphilis and the possibilities of various combinations are discussed.

The author finishes with a reminder of our ignorance of the essence of the paralytic process; in this context he refers to the puzzle of his two rabbits and one dog, which presented in their cortex the typical histopathological picture of general paralysis.

The book, containing the results of two such accurate observers as Alzheimer and Nissl, must be regarded as the most complete and trustworthy presentation of the histopathology of general paralysis hitherto published. The ideal of histological analysis accepted by the editor involves the careful scrutiny with the oil-immersion of every cell and tissue element, and a clear appreciation of the extent of one's knowledge of each element. Such an ideal involves an amount of detailed labour which only a

strong scientific faith makes possible, and it is good to find that the result of this long-continued research is a contribution to the subject so stimulating in its presentation, and so far-reaching in its significance.

The editor is to be congratulated on having had the co-operation of a publisher who has done justice to the work.

C. MACFIE CAMPBELL.

**AFECTIVITÄT, SUGGESTIBILITÄT, PARANOIA.** E. BLEULER,  
Prof. of Psychiatry, Zürich. Halle a. S.: Carl Marhold. 1906.  
Pp. 144.

THE great importance of the affective side of all mental operations, both in normal and pathological psychology, has of late years received increasing recognition, and the above very thorough analysis of affective states with particular reference to paranoia from the able pen of Professor Bleuler is a valuable addition to the literature of psychiatry. The author sharply distinguishes affectivity (emotion—feeling—affection) from intellectual processes, whilst he admits that the separation is purely a theoretical one, as in actuality every intellectual state or process has its emotional complement. Certain intellectual states, however, have particular connection with affectivity, *e.g.* sensations of muscular tension, palpitation, hunger, thirst, etc. As regards the vexed question of pain (*Schmerz*, not *Unlust*), the author himself is not clear whether it should be regarded as a sensation or a feeling. Probably both. However this may be, he emphasises the preponderating importance of affectivity in action, as it “generalises the reaction to isolated sensory impressions over the whole body and mind, pushes opposing tendencies out of the way, and decides the range and force of the reaction.” It is thus the dominating motive power in conduct, and is, further, in pathological or abnormal conditions, the source of dissociations and transformations of the personality, and the origin of certain forms of delirium. Affectivity still further displays a well-defined independence as contrasted with intellectual processes, the affective complement of one succeeding state being frequently carried over to succeeding mental operations. This affective solidarity is also evidenced by the fact that in childhood the development of affectivity is independent of, and quite out of proportion to, that of the intellect, so that in pathological development either the intellectual idiot or the emotional or moral idiot may result. In this relation the author adverts to the advantage of classification of individual types, as attempted by the old divisions into temperaments, sanguine, phlegmatic, and so on. Attention is next analysed and finally defined as a special

form of affectivity. In pathological states, affectivity, the author says, dominates the whole clinical picture. In the organic psychoses, the emotions are, he maintains, not obliterated as is commonly affirmed, their elimination being apparent, not real, depending entirely on loss of exciting intellectual elements. Similarly with alcoholics and epileptics the emotional elements persist but are extraordinarily labile, producing a factitious effacement. In idiocy, all variations of affectivity are to be found, just as in the normal, only with a much wider range, but in dementia præcox the affective reactions are largely suppressed. Turning to suggestibility, Professor Bleuler shows that in result, in their mode of operation, and in origin, affectivity and suggestion are identical, suggestion being an affective process and suggestibility merely one phase of affectivity. The importance of this contention in its bearing on the phenomena of hysteria and hypnotic suggestion is obvious. All these considerations naturally lead up to the author's examination of the mechanism of paranoia. Concerning the ground-work of paranoia, and whether in this category many different conditions are united or not, we are, Professor Bleuler maintains, still in the dark, but he is convinced, and his arguments go far to prove, that it is not derived from pathologically altered affective states, as is widely held. In particular, morbid suspicion—one of the most constant symptoms—is not an "affect," but is only a secondary effect of intellectual disorders (delusions, etc.). Neither is there in paranoia a general disturbance of perception or apperception or of memorial images. Professor Bleuler devotes considerable space to the discussion of the marked egotism (*Hyper-tropie des Ich, egozentrischer Charakter*) so frequently observed in paranoiacs. None of these features are constant, and even when present are operative only in a numerically limited part of the experiences of the subject, and are not spread over the whole field as they would be if of primarily affective origin. The ego-centric character of the paranoiac is thus merely the result of the circumstance that in the foreground of his mind there is continually present an affectively-accentuated idea complex; his delusions being intellectually and not affectively determined, differing from the mistakes of the sane only by their incorrigibility. Herein, however, lies the crux of the whole matter, and though Professor Bleuler says that this characteristic incorrigibility may rest on chemical, anatomical, or "functional" grounds, his explanation throws no light on this supremely important point. The whole question is highly controversial and some of the author's conclusions, particularly in this last chapter on paranoia, can only be accepted with considerable reservation. Nevertheless the book as a whole will be found in many directions illuminating and in others highly suggestive.

R. CUNYNGHAM BROWN.

# Bibliography

## ANATOMY

- VAN GEHUCHTEN. L'Anatomie du Système Nerveux. 4me édition, remaniée et augmentée. Uystpruyst, Louvain, 1906, 30 fr.
- EDUARD PFLÜGER. Ueber den elementaren Bau des Nervensystems. *Arch. f. Physiol.*, Bd. 112, H. 1, p. 1.
- ENRICO ROSSI. Ulteriori ricerche sulla intima struttura delle cellule nervose nei vertebrati. *Nervaze*, Vol. vii., F. 3, 1906, p. 327.
- SCHIEFFERDECKER. Über das Verhalten der Fibrillen des Achsenzylinders an den Ranvierschen Einschnürungen der merkhaltigen Nervenfasern. *Archiv. f. mik. Anat.*, 1906, p. 783.
- PARHON et NADEJDE. Nouvelle contribution à l'étude des localisations dans les noyaux des nerfs craniens et rachidiens chez l'homme et chez le chien. *Journ. de Neurol.*, avril 5, 1906, p. 129.
- SYMINGTON. A Note on the Topographical Anatomy of the Caput Gyri Hippocampi. *Journ. of Anat. and Physiol.*, Vol. xl., Part iii., p. 244.
- J. T. WILSON. On the Anatomy of the Calamus region in the Human Bulb; with an account of a hitherto undescribed "Nucleus Postvermis." Part i. *Journ. of Anat. and Physiol.*, Vol. xl., Part iii., p. 210.
- BERTHE DE VRIESE. Sur la signification morphologique des artères cérébrales. *Arch. de Biol.*, T. xxi., F. iii. et iv., 1905, p. 357.
- KAMON. Zur Entwicklungsgeschichte des Gehirns des Hühnchens. *Anat. Hefte*, H. 92 (Bd. 30, H. 3), p. 559.
- ANDRÉ THOMAS. Application de la méthode de Ramon y Cajal (impregnation à l'argent) à l'anatomie pathologique du cylindrace. *Rev. Neurol.*, mars 31, 1906, p. 249.
- IRVING HARDESTY. A Class Model of the Spinal Cord. *Bull. Johns Hopkins Hosp.*, Feb. 1906, p. 43.

## PHYSIOLOGY

- MORAT. Physiology of the Nervous System. Constable, New York, 1906. Translated by H. W. Syers. Price 31s. 6d.
- PATRIZI. Sur quelques points controversés de la physiologie du cervelet. *Arch. Ital. de Biol.*, T. xlv., F. 1, 1906, p. 18.
- L. P. BARKER. The Neurones (concluded). *Journ. of Am. Med. Ass.*, April 7, p. 1006.
- ZANIEWSKI. Sur l'analogie et l'identité des lois modernes de l'excitation avec mes expériences cliniques antérieures. *Ann. d'Electrobiol. et de Radiol.*, mars 1906, p. 145.
- BERTHOLET. Les voies de la sensibilité douloureuse et calorifique dans la moelle. *Nervaze*, Vol. vii., F. 3, 1906, p. 283.
- KOPCZYNSKI. Experimentelle Untersuchungen aus dem Gebiete der Anatomie und Physiologie der hinteren Spinalwurzeln. *Neurol. Centralbl.*, April 1, 1906, S. 297.
- TRICOMI-ALLEGRA. Studio sperimentale sulla via acustica fondamentale. *Nervaze*, Vol. vii., F. 3, 1906, p. 227.
- RAMSTRÖM. Untersuchungen über die Nerven des Diaphragma. *Anat. Hefte*, H. 92 (Bd. 30, H. 3), p. 669.
- DEVAUX. Théorie osmotique du sommeil. *Arch. gén. de méd.*, avril 10, 1906, p. 903.
- SERGI. Le système nerveux central dans les mouvements de la testudo græca. *Arch. Ital. de Biol.*, Vol. xlv., 1906, p. 90.

## PSYCHOLOGY

- GOLDSTEIN. Merkfähigkeit, Gedächtnis und Assoziation (Schluss). *Zeit. f. Psych. u. Phys. der Sinnesorgane*, Bd. 41, H. 2 u. 3, 1906, p. 117.
- A. O. LOVEJOY. Kant's Antithesis of Dogmatism and Criticism. *Mind*, April, p. 191.
- W. H. WINCH. Psychology and Philosophy of Play (II.). *Mind*, April, p. 177.
- F. C. S. SCHILLER. The Ambiguity of Truth. *Mind*, April, p. 161.



- NORMAN SMITH. Avenarius' Philosophy of Pure Experience. *Mind*, April, p. 149.
- MAX TOTH. Wie rahmen wir unsere Bilder ein? *Zeit. f. Psych. u. Phys. der Sinnesorgane*, Bd. 41, H. 2 u. 3, 1906, p. 145.
- LOUP. Les Idées de négation dans les états hypochondriaques. *Thèse*. Rey et Cie, Lyon, 1906, 2 fr.
- GRASSET. Le Psychisme inférieur (étude de physiopathologie clinique des centres psychiques). Chevalier et Rivière, Paris, 1906, 9 fr.
- ADAMKIEWICZ. Pensée inconsciente et Vision de la pensée. Essai d'une explication physiologique du processus de la pensée et de quelques phénomènes "surnaturels" et psychopathiques. Roussel, Paris, 1906, 2 fr.
- MORTON PRINCE. The Psychology of Sudden Religious Conversion. *Journ. Abnormal Psychol.*, Vol. i., No. 1, 1906, p. 42.
- FÈRE. Deuxième note sur la fausse réminiscence. *Journ. de Neurol.*, mars 20, 1906, p. 101.
- RIVERS. Report on the Psychology and Sociology of the Todas and other Indian Tribes. *Proc. Royal Soc.*, No. B. 517, Feb. 26, 1906, p. 239.
- BECHTEREW. What is Hypnosis? *Journ. Abnormal Psychol.*, Vol. i., No. 1, 1906, p. 18.
- HAUGG. Theorie und Praktik der Hypnose und Suggestion, Hypnotismus. Scherzer, München, 1906, M. 1.20.

### PATHOLOGY

- MARINESCO et MINEA. Recherches sur la régénérescence des nerfs périphériques. *Rev. Neurol.*, avril 15, 1906, p. 301.
- BURZIO. Contributo allo Studio Anatomo-istologico del Sistema Nervoso nel Cretinismo. *Ann. di Freniatria*, Vol. xvi., F. 1, 1906, p. 32.
- BIANCHINI. Intorno alla degenerazione e all rigenerazione dei nervi. *La clinica moderna*. Feb. 19 and 28, pp. 85 and 97.
- NAGER. Über postmortale histologische Artefakte am N. acusticus und ihre Erklärung, ein Beitrag zur Lehre der Corpora amylacea. *Ztschr. f. Nervenheilk.*, Bd. 51, H. 3, p. 250.
- SOPRANA. Examen microscopique du système nerveux et du système musculaire d'un pigeon chez lequel l'ablation des canaux semicirculaires avait été suivie d'une très grave atrophie musculaire. *Arch. ital. de Biol.*, Vol. xlv., p. 135.

### CLINICAL NEUROLOGY AND PSYCHIATRY

#### GENERAL—

- SMITH ELY JELLIFFE. Dispensary Work in Nervous and Mental Diseases. *Journ. Nerv. and Ment. Dis.*, April 1906, p. 234.
- RIGNANO. Sur la transmissibilité des caractères acquis. Hypothèse d'une centro-épigénèse. Felix Alcan, Paris, 1906.
- LILIENSTERN. Beitrag zur Lehre vom Aufbrauch durch Hyperfunktion. *Münch. med. Wchnschr.*, April 17, p. 748.
- ANTON. Über den Wiederersatz der Funktion bei Erkrankungen des Gehirns. S. Karger, Berlin, 1906, M.—80.
- ALFRED MANTLE. The Influence of the Nervous System and External Temperature upon certain Circulatory Changes concerned in the Etiology of Catarrh, Ulcer, and Simple Dilatation of the Stomach. *Lancet*, April 14, 1906, p. 1031.
- v. LEYDEN. Röntgenstrahlen und innere Medizin insbesondere Wirbel und Rückenmarkserkrankungen. *Zeit. f. physikal. u. diätet. Therapie*, April 1, 1906, p. 5.
- LEMOYNE. Contribution à l'étude des troubles mentaux dans la chlorose. *Thèse*. Schneider, Lyon, 1905.

#### MUSCLES—

- LEUBUSCHER. Über einen Wahrscheinlich durch Bleivergiftung entstandenen Fall von Myoklonie, April, p. 347.
- FONTANEL. Nosographie et Physiologie pathologique de l'asthénie motrice bulbo-spinale, syndrome d'Erb-Goldflam. Schneider, Lyon, 1906.
- CAMUS. Les Amyotrophies myélopathiques à type Aran-Duchenne d'origine syphilitique. Schneider, Lyon, 1906.

#### PERIPHERAL NERVES—

- JAMES SHERREN. The Erasmus Wilson Lectures on the Distribution and Recovery of Peripheral Nerves studied from instances of Division in Man. Lecture III. *Lancet*, March 31, 1906, p. 886.
- BRIFFAUT. Contribution à l'étude des polynévrites tuberculeuses (maladie de Landry). Schneider, Lyon, 1906.

- DUNGER. Ueber uraemische Neuritis. *Münch. med. Wchnschr.*, April 17, p. 745.  
 FREDERICK PRICE. A Case of Widespread Motor Paralysis due to Multiple Symmetrical Peripheral Neuritis. *Brit. Med. Journ.*, April 21, 1906, p. 914.  
 DUBRUEL. Le Beri-beri. Baillière et fils, Paris, 1906, 3s. 6d.  
 BRISSAUD et MOUTIER. Les paralysies de l'épaule à volonté et la loi sur les accidents du travail. *Presse méd.*, avril 25, p. 250.  
 ESPOSITO. Amiotrofia da trauma nervoso periferico. Studio clinico (cont.). *Manicomio*, Anno xxi, No. 3, 1905, p. 350.

## SPINAL CORD—

- Poliomyelitis Anterior Acuta.**—WICKMAN. Studien über Poliomyelitis acuta. S. Karger, Berlin, 1906, 6s.  
 TREVELYAN. On fifty cases of Infantile Paralysis. *British Journ. of Children's Diseases*, April 1906, p. 135.  
**Tabes.**—DAVID FERRIER. The Lumleian Lectures on Tabes Dorsalis. *Brit. Med. Journ.*, March 31, April 7 and 14. *Lancet*, March 31, April 7 and 14, 1906.  
 MARINESCO. Contribution à l'étude de l'histologie et de la pathogénie du tabes. *Semaine Méd.*, avril 18, p. 181.  
 SCHÜLLER. Über atypischer Verlaufsformen der Tabes. *Wien. med. Woch.*, April 14, p. 762, April 21, p. 818 (Fortsetzung folgt).  
 DE REZENDE PUECH. Tabes dorsalis e Tabes associada. *Arch. Brasil. de Psychiat. e Neurol.*, Anno ii., No. 1, 1906, p. 58.  
 ANNA AVDAKOFF. La Paralysie de la branche external du Spinal dans le Tabes. Rousset, Paris, 1906.  
 BELUGOU. Les traitements utiles du Tabes. *Arch. gén. de méd.*, avril 3, 10, et 17, 1906, pp. 851-914.  
 DREVON. Contribution à l'étude du traitement du tabes par les injections de calomel. *Thèse*. Schneider, Lyon, 1906.  
**Myelomalacia.**—SCHLAPP. A Case of Ascending Myelomalacia caused by a Progressing Venous Thrombosis. *New York Med. Journ.*, April 7, p. 694.  
**Syringomyelia.**—RAYMOND et FRANÇAIS. Note sur un cas de syringomyélie avec troubles sensitifs à topographie radiculaire. *Rev. Neurol.*, mars 31, 1906, p. 253.  
 HERBERT C. MOFFITT. Leprosy Simulating Syringomyelia. *Journ. Nerv. and Ment. Dis.*, April 1906, p. 249.  
**Disseminated Sclerosis.**—SELLING. Main de prédicateur bei multipler Sklerose. *Münch. med. Woch.*, April 24, p. 801.  
**Cerebro-spinal Fluid.**—MERZBACHER. Die Beziehung der Syphilis zur Lymphocytose der Cerebrospinalflüssigkeit und zur Lehre von der "meningitischen Reizung." *Centralbl. f. Nervenheilk. u. Psychiat.*, April 15, 1906, S. 304.

## BRAIN—

- ANGELO PIAZZA. Contributo clinico ed anatomo-patologico alle lesioni del nucleo lenticolare. *Riv. di Patol. nerv. e ment.*, Vol. xi., F. 2, 1906, p. 73.  
**Meningitis.**—CORKHILL. A Case of Posterior Basal Meningitis with Few Cerebral Symptoms. *Brit. Med. Journ.*, March 31, 1906, p. 734.  
 KNAPP. Serous Meningitis. *Arch. of Otol.*, Vol. xxxv., No. 1, p. 1.  
 HILDESCHHEIM. The Prognosis in Posterior Basic Meningitis. *Brit. Med. Journ.*, March 31, 1906, p. 733.  
 KOLLE u. WASSERMANN. Versuche zur Gewinnung und Wertbestimmung eines Meningococcenserums. *Deutsche med. Woch.*, April 19, p. 609.  
 HALL and HOPKINS. Tuberculous Meningitis, with Report of 52 Cases. *Journ. Nerv. and Ment. Dis.*, April 1906, p. 242.  
 VILLARET et TIXIER. Certaines formes de méningite tuberculeuse. *Rev. de la Tuberculose*, fév. 1906, p. 1 (à suivre).  
 E. L. DOW. The Treatment of Meningococcus. *Med. Rec.*, March 31, p. 495.  
**Hæmorrhage.**—ROTKY. Ein Fall von akuter Phosphorvergiftung mit Hirn Hæmorrhagie. *Prag. med. Woch.*, April 26, p. 219.  
**Sinus Thrombosis.**—DUNDAS GRANT. Two Cases of Otitic Lateral Sinus Phlebitis, one complicated with Cerebellar Abscess. *Lancet*, April 14, 1906, p. 1039.  
 CARRIÈRE et VERMESCH. Thrombo-phlébite post-grippale du sinus. *Arch. gén. de méd.*, avril 3, 1906, p. 846.  
**Tumour.**—KNAPP. The Mental Symptoms of Cerebral Tumour. *Boston Med. and Surg. Journ.*, April 5, p. 361.  
 SCHORSTEIN and WALTON. A Case of Glioma of the Pons. *Lancet*, April 14, 1906, p. 1035.  
 I. W. BLACKBURN and W. H. HOUGH. Endothelial Spindle-celled Sarcoma of the Dura Mater penetrating the Brain. *New York Med. Journ.*, April 7, p. 689.

- Hydatid.**—H. EGERTON BROWN. A Case of *Cysticercus Cellulose* occurring in the Insane. *Rev. Neurol. and Psychiat.*, April 1906, p. 272.  
 SANDRI. Un caso di echinococco multiplo del cervello. *Riv. di Patol. nerv. e ment.*, Vol. xi., f. 2, 1906, p. 61.  
**General Paralysis.**—W. FORD ROBERTSON. The Pathology of General Paralysis of the Insane. *Rev. Neurol. and Psychiat.*, April 1906, p. 259.  
 BOUVAIST. La Paralyse générale dans l'Aveyron. Contribution à l'étude de l'étiologie de la paralysie générale progressive. *Thèse*. Firmin, Montane et Sicardi, Montpellier, 1906.  
 FODERÉ. Névroses et Paralysie générale. Leclerc, Paris, 1906.  
 CH. PÉRÉ. L'angoisse au cours de la paralysie générale. *Rev. de Méd.*, avril 10, 1906, p. 329.  
**Little's Disease.**—BOUCHAUD. Paraplégie infantile. Début insidieux, état stationnaire, puis aggravation. *Journ. de Neurol.*, mars 20, 1906, p. 105.  
**Pseudobulbar Paralysis.**—HOERSLIN u. SELLING. Beitrag zur Kenntnis der Pseudobulbärparalyse. *Münch. med. Woch.*, April 24, p. 799.  
**Cerebellar Ataxia.**—ABRUZZETTI. Contributo allo studio clinico dell' atassie cerebellare (continua). *Riv. Crit. di Clin. Med.*, April 21, p. 245.

#### MENTAL DISEASES—

- ILBERG. Bericht über die ersten 100 Sitzungen der forensisch-psychiatrischen Vereinigung zu Dresden. Marhold, Halle, 1906.  
 TUCZEK. Die wissenschaftliche Stellung der Psychiatrie. Elwert's Verlag, Marburg, 1906, M.—50.  
 BUNNEMANN. Ueber den Begriff des Psychischen. *Centralbl. f. Nervenheilk. u. Psychiat.*, April 15, 1906, S. 239.  
 INGEGNIEROS. Nuova classificazione dei delinquenti fondata sulla psicopatologia. *Manicomio*, Anno xxi., No. 3, 1905, p. 311.  
 PINI. Le cause delle recidive nelle alienazioni mentali. *Manicomio*, Anno xxi., No. 3, p. 273.  
 WILLIAM A. WHITE. Types in Mental Diseases. *Journ. Nerv. and Ment. Dis.*, April 1906, p. 254.  
 GEORGE STOCKTON. The Importance of the Early Diagnosis of Mental Diseases. *Journ. Nerv. and Ment. Dis.*, April 1906, p. 265.  
 AUSTREGESILLO. Mimotismo nos imbecis e idiotas. *Arch. Brasil. de Psychiat. e Neurol.*, Anno ii., N. 1, 1906, p. 3.  
 FRANCESCHI. Un caso di distimia ciclica a fasi brevissime in una imbecille grave. *Riv. di Patol. nerv. e ment.*, Vol. xi., f. 2, 1906, p. 49.  
 PENTA. Die Simulation von Geisteskrankheiten. Stubor's Verlag, Würzburg, 1906, M. 3.60.  
 PIERRE JANET. The Pathogenesis of Some Impulsions. *Journ. Abnormal Psychol.*, Vol. i., No. 1, 1906, p. 1.  
 BUMKE. Was sind Zwangsvorgänge? Carl Marhold, Halle a. S., 1906, M. 1.20.  
 HUDOVERNIG. Ein Fall von peripher entstandener Sinnestäuschung. *Centralbl. f. Nervenheilk. u. Psychiat.*, April 1, 1906, S. 255.  
 U. V. FILHO. Contribuição ao estudo das estereotypias. *Arch. Brasil. de Psychiat. e Neurol.*, Anno ii., No. 1, 1906, p. 30.  
 CHARLES MERCIER. Folie à Deux (?). *Brit. Med. Journ.*, March 31, 1906, p. 735.  
 ARSIMOLES. Deux cas de Mélancholie anxieuse. Sitophobie intermittente à périodicité régulière. Double personnalité coexistante. *Arch. gén. de méd.*, mars 7, 1906, p. 790.  
 DEL GRECO. Intorno alla mente ed ai suoi tipi. Saggio di psicologia concreta. *Manicomio*, Anno xxi., No. 3, 1905, p. 321.  
 A. MARIE. La démence. Octave Doin, Paris, 1906, 4 fr.  
 BESSIERE. Les stéréotypies démentielles. *Ann. Méd.-Psych.*, mars-avril, p. 206.  
 GIMBAL. Les incendiaires (suite). *Ann. Méd.-Psych.*, mars-avril, p. 214.  
 TOBBEN. Ein Beitrag zur Kenntnis des Eifersuchtswahns. *Monatssch. f. Psychiat. u. Neurol.*, April, p. 321.  
 TOMASINI. Sull' agitazione degli alienati e la cure morfioscopolaminica. *Manicomio*, Anno xxi., No. 3, 1905, p. 290.  
 SOUCHANOFF. Les représentations obsédantes hallucinations et les hallucinations obsédantes. *Rev. de Méd.*, avril 10, 1906, p. 336.  
 CRISTIANI. Frenastenia passiva di violenza carnale. *Manicomio*, Anno xxi., No. 3, 1905, p. 343.  
 CROCQ. Les formes frustes de la démence précoce. *Journ. de Neurol.*, avril 5, 1906, p. 121.

- MOREIRA.** Psychose em leprosos. *Arch. Brasil. de Psychiat. e Neurol.*, Anno ii, N. 1, 1906, p. 41.
- MÜNZER.** Ein Beitrag zur Lehre der Puerperalpsychosen. *Monatsch. f. Psychiat. u. Neurol.*, April, p. 362.
- NINA-RODRIGUES.** La psychose polynévritique et le bériberi. *Ann. Méd.-Psychol.*, mars-avril, p. 177.
- KNAPP.** Les Psychoses Polynévritiques. Bergmann, Wiesbaden, 1906.
- GREGOR und ROEMER.** Zur Kenntnis der Auffassung einfacher optischer Sinneseindrücke bei alkoholischen Geistesstörungen, insbesondere bei der Korsakoff'schen Psychose. *Neurol. Centralbl.*, April 16, 1906, S. 339.
- BACCELLI.** Otoematoma nei malati di mente. *Manicomio*, Anno xxi., No. 3, 1905, p. 372.
- TIRELLI.** Perizia medico-legale sulle condizioni mentali di Rosa Bonetti. *Ann. di Freniatria*, Vol. xvi., F. 1, 1906, p. 1.
- COURBON.** Étude psychiatrique sur Benvenuto Cellini. *Thèse.* Maloine, Paris, 1906.
- LACOMBE.** La Psychologie des individus et des sociétés chez Taine. F. Alcan, Paris, 1906, 7 fr. 50.
- STEWART PATON.** The Care of the Insane and the Study of Psychiatry in Germany. *Journ. Nerv. and Ment. Dis.*, April 1906, p. 225.
- JULIANO MOREIRA.** Ligeiras notas a proposito da Assistencia familiar. *Arch. Brasil. de Psychiat. e Neurol.*, Anno ii., N. 1, 1906, p. 25.
- FRANCO DA ROCHA.** Assistencia familiar aos alienados em S. Paulo. *Arch. Brasil. de Psychiat. e Neurol.*, Anno ii., N. 1, 1906, p. 18.
- Alcohol.**—**PFÄFF.** Die Alkoholfrage vom ärztlichen Standpunkt. Reinhardt, München, 1906, M. 1.60.
- BARON.** De l'alcoolisme acquis du nourrisson et de l'enfant. Schneider, Lyon, 1906.
- CHARRA.** Contribution à l'étude de l'alcoolisme héréditaire. Recherches sur les anomalies de développement observées chez les enfants de parents alcooliques. *Thèse.* Ret, Lyon, 1906, 2 fr.

## GENERAL AND FUNCTIONAL DISEASES—

- Chorea.**—**SIR W. R. GOWERS.** On Tetanoid Chorea and its Association with Cirrhosis of the Liver. *Rev. Neurol. and Psychiat.*, April 1906, p. 249.
- A. A. ESHNER.** Chorea and some disorders simulating it. *Med. Rec.*, April 7, 1906, p. 547.
- Epilepsy.**—**SCHUCKMANN.** Kritisches und experimentelles zur Toxinhypothese der Epilepsie. *Monatsch. f. Psychiat. u. Neurol.*, April, p. 332.
- W. ALDREN TURNER.** Dietetic Treatment in Epilepsy. *Practitioner*, April 1906, p. 553.
- A. M'DOUGALL.** A Case of Senile Epilepsy treated with potassium iodide. *Med. Chronicle*, April, p. 12.
- EDWIN BRAMWELL.** The Sane Epileptic and the Colony System. *Scot. Med. and Surg. Journ.*, April 1906, p. 331.
- D. P. ALLAN, H. L. SANFORD, and D. H. DOLLEY.** Traumatic Defects of the Skull; their Relation to Epilepsy. Boston. *Med. and Surg. Journ.*, April 12, p. 396.
- Hysteria.**—**R. VOGT.** Die hysterischen Dissoziationen im Lichte der Lehre von der Energie-Absorption. *Centralbl. f. Nervenheilk. u. Psychiat.*, April 1, 1906, S. 249.
- INGEGNIEROS.** Sul linguaggio musicale negl'isterici. *Manicomio*, Anno xxi., No. 3, 1905, p. 303.
- MATHIEU et ROUX.** Des Hématémèses hystériques. *Gaz. des Hôp.*, avril 26, p. 565.
- BUMKE.** Ueber Pupillenstarre im hysterischen Anfall. *Münch. med. Woch.*, April 17, p. 741.
- LÖWMAN.** Angio-neurotic Oedema. *Lancet*, April 14, 1906, p. 358.
- PUTNAM.** Recent Experiences in the Study and Treatment of Hysteria at the Massachusetts General Hospital; with Remarks on Freud's Method of Treatment by "Psycho-Analysis." *Journ. Abnormal Psychol.*, Vol. i., No. 1, 1906, p. 26.
- Neurasthenia.**—**CONVERS.** Psychoses et Neurasthénies en rapport avec les maladies du nez et du rhino-pharynx. Lengendre et Cie, Lyon, 1906.
- WOLLENBERG.** Ueber das psychische Moment bei der Neurasthenie. *Deutsche med. Woch.*, April 26, p. 659.
- GAUCKLER.** Quelques considérations sur le traitement des neurasthéniques. Steinheil, Paris, 1906.

**Traumatic Neuroses.**—BIONDI. Sulla Sintomatologia e sulla Patogenesi delle neurosi traumatiche. *Ann. di Freniatria*, Vol. xvi., F. 1, 1906, p. 52.

**Exophthalmic Goitre.**—HENRY TEMPEST DUFTON. A Case of Exophthalmic Goitre, with Remarks on the Probable Nature of the Disease. *Brit. Med. Journ.*, April 21, 1906, p. 914.

ROBINSON. On the Relationship between Grave's Disease and Acute Rheumatism. *Lancet*, April 14, 1906, p. 1037.

FREUDENBERG. Ein Fall von Morbus Basedow, behandelt mit Antithyreoidin. Koenig, Leipzig, 1906, M. 1.

STEGMANN. Bemerkungen zur Behandlung des Morbus Basedowii mit Röntgenstrahlen. *Wien. klin. Woch.*, April 19, p. 473.

E. F. CURTIS. The Results of the Surgical Treatment of Exophthalmic Goitre. *Annals of Surgery*, March 1906, p. 335.

**Acromegaly.**—WIDEL, ROY, et FROIN. Un cas d'acromégalie sans hypertrophie du corps pituitaire avec formation kystique dans la glande. *Rev. de Méd.*, avril 10, 1906, p. 313.

#### SPECIAL SENSES AND CRANIAL NERVES—

AXENFELD. Ueber traumatische reflectorische Pupillenstarre. *Deutsche med. Woch.*, April 26, p. 663.

DREYFUS. Ueber traumatische Pupillenstarre. *Münch. med. Woch.*, März 27, p. 604.

PFLUGK. Über die Akkomodation des Auges der Taube nebst Bemerkungen über die Akkomodation des Affen (*Macacus cynomolgus*) und des Menschen. Bergmann, Wiesbaden, 1906, M. 3.60.

CANTONNET et TAGUET. Paralyse des mouvements associés des yeux et leur dissociation dans les mouvements volontaires et automatico-réflexes. *Rev. Neurol.*, avril 15, 1906, p. 308.

WILHELM SCHOEN. Das Schielen, Ursachen, Folgen, Behandlung. Lehmanns Verlag, München, 1906.

HASTINGS. A case of acute middle ear suppuration complicated by labyrinthine fistula and paralysis of the abducens nerve. *Arch. of Otol.*, Vol. xxxv., No. 1, p. 1.

JOHMANN. Rezidivierende Oculomotoriuslähmung als Komplikation bei Typhus abdominalis. *Deutsche med. Woch.*, April 19, p. 617.

L. STREET. Motor ocular paralysis as a complication of acute articular rheumatism. *New York Med. Journ.*, April 7, p. 712.

DUMAREST. Des névroses et névrites du pneumogastrique chez les tuberculeux. Gainche, Paris, 1906.

#### MISCELLANEOUS SYMPTOMS—

LENNANDER. Ueber lokale Anaesthesia und über Sensibilität der Organe und Gewebe, weitere Beobachtungen. II. *Mitteil a. d. Grenzgeb.*, Bd. 15, H. 5, p. 465.

KEMPNER. Ueber Störungen im Augengebiet des Trigemini speziell des Cornealreflexes und ihre diagnostische Verwertung. *Berlin. klin. Woch.*, März 26, p. 379.

GOLDSCHIEDER. Kasuistischer Beitrag zur Lehre von der sensorischen Ataxie. *Neurol. Centralbl.*, April 16, 1906, S. 338.

BAUMANN. Ueber den Rachenreflex. *Münch. med. Woch.*, März 27, p. 593.

KURT MENDEL. Über den Fussrückenreflex. *Neurol. Centralbl.*, April 1, 1906 S. 293.

LASAREW. Über den Schäfer'schen antagonistischen Reflex. *Neurol. Centralbl.*, April 1, 1906, S. 291.

BÉCHTEREW. Über eine eigentümliche Reflexerscheinung bei Plantarflexion des Fusses und der Zehen in Fällen von Affektion des centralen motorischen Neurons. *Neurol. Centralbl.*, April 1, 1906, S. 290.

LOEB. Gutachten über Entstehung und Rückbildung traumatischer Aphasie. *Mitteil a. d. Grenzgeb.*, Bd. 15, H. 5, p. 495.

#### TREATMENT\*—

ERIC CAMPBELL PRITCHARD. The Training of Nerve Centres in Children. *Lancet*, April 21, 1906, p. 1104.

GUTZMANN. Grundzüge der Behandlung nervöser Sprachstörungen. *Deutsche med. Woch.*, April 19, p. 612.

BIENFAIT. Le traitement des dyspepsies nerveuses et des troubles nerveux dyspeptiques. *Ann. d'Électrobiol. et de Radiol.*, mars 1906, p. 173.

MORÉL. Sur les lésions du sinus latéral et leur traitement longitudinal dans es traumatismes crâniens. *Arch. gén. de méd.*, avril 24, 1906, p. 104.

PERS. Ueber chirurgische Behandlung der Ischies. *Deutsche med. Woch.* April 12, p. 574.

\* A number of references to papers on Treatment are included in the Bibliography under the individual Diseases.

# Review of Neurology and Psychiatry

---

## Original Articles

### THE PATHOLOGY OF A CASE OF MYELITIS ACUTISSIMA HÆMORRHAGICA DISSEMINATA

By W. B. WARRINGTON, M.D., F.R.C.P.,

and

JOHN OWEN, M.B.

(From the Thompson-Yates Laboratories of the University of Liverpool.)

IN the *Liverpool Medico-Chirurgical Journal* for June 1904, Mr George Hamilton recorded a case of rapid paraplegia terminating fatally in four and a half days. The patient, a gentleman æt. 41, was under the care of Mr Hamilton, and seen in consultation by Dr T. R. Glynn and the late Dr Alexander Davidson.

The following are the essential clinical features :—

No history of previous illness was obtained. On October 27th, 1904, the patient took a "chill," and felt out of sorts; he was, however, able to travel up to London. Three days later Mr Hamilton found him with a temperature of 101° and symptoms of slight gastric catarrh. He rapidly improved, and on the 4th November seemed quite well. On the afternoon of that day, when standing by his dressing-table, he said suddenly, "I think I have lost the use of my legs," and before he could be got into bed he was completely paralysed from the waist down. Mr Hamilton found complete motor and sensory paralysis below the umbilicus. The reflexes were absent, and there was retention

of urine. The same evening there was violent pain in the spine at the mid-dorsal region. The following day loss of sensation extended to a higher level; at midnight he became unconscious, breathing purely with the diaphragm. The left pupil was larger than the right, and both reacted to light. There was much difficulty in swallowing. Paralysis of the upper limbs and of the right side of the face soon followed. The temperature rose suddenly on the afternoon of the illness, and remained high until death four and a half days after the onset.

An autopsy was made 24 hours after death by Dr Ernest Glynn, who found no disease in the viscera, the spleen was slightly enlarged, and its substance diffuent; from it unsuccessful attempts were made to cultivate micro-organisms. The brain and spinal cord were hardened in formalin, and after some preliminary examination Dr Glynn kindly handed the material over to us for a further investigation.

On cutting across the cord at various levels, masses of intraspinal hæmorrhage were readily seen with the naked eye. The maximum intensity of the hæmorrhage was found at the lower dorsal region, where the greater part of the cord appeared to be replaced by a clot; from here to the termination of the cord there was much hæmorrhage. The next chief site was in the mid-cervical region. Smaller extravasations were seen scattered throughout the cord and extending as high as the upper part of the medulla. The membranes appeared normal, the pia being smooth and glistening, and showing through it the greatly dilated spinal arteries and veins.

The microscopical findings may be briefly described under the following headings:—

Distribution of the hæmorrhage and vascular changes.

Changes in the vessel walls and their immediate neighbourhood.

Swelling and necrosis of tissue.

Condition of the nerve elements.

1. *Distribution of the Hæmorrhage and Vascular Changes.*—

The figures 1 and 2 are drawn from low power magnification with the Edinger projection apparatus, and reproduce the appearance seen in sections taken from the lumbar and dorsal regions, and stained by van Gieson's method. Their inspection shows that

the hæmorrhage followed chiefly the distribution of the anterior spinal system of arteries, but that the coronal vessels had in many places ruptured. The extravasation of blood and fluid in the lumbar region caused a marked dislocation of the ventral cornua towards the periphery, and in the cervical region had torn across the base of one posterior cornu at its attachment to the central grey. Apart from this destruction of tissue caused by the irruption of blood from the branches of the anterior spinal system, a distinctive feature shown by the sections was the great dilatation of innumerable small vessels, not merely in the grey matter, but scattered over the conducting tracts of the cord. In the van Gieson specimens these vessels and the infiltrated surrounding tissue stood out as brilliant red disseminated areas. Where the hæmorrhage was copious, numerous granules of blood pigments were seen, much of which was enclosed in large, round, clear cells. In the pia there were greatly distended arteries and veins, the latter containing many pigment granules and surrounded by round mononuclear cells; the vessels in the nerve roots also in many instances were distended, in other places ruptured.

2. *Changes in the Vessel Walls and their Neighbourhood.*—

The structure of the larger vessels was normal, there was no thickening of their walls, and the acid orcein stain showed the normal elastica without change in the internal coat; but both in the pia and around the greatly dilated branches of the anterior spinal arteries there was infiltration of the surrounding tissue with round and fusiform mononuclear cells. In the central grey, when vessels cut in transverse sections could be seen, the perivascular sheaths were found filled with blood cells and round cells which also infiltrated the neighbouring tissues. In others, as in Fig. 3, the lumen of the vessel was bounded by granular necrotic masses, in which only few formed elements could be detected, replacing the vessel wall and perivascular space. Outside this again was another zone of necrotic tissue. Changes in the smaller vessels, on the other hand, presented a characteristic feature of the histological study of the cord. The bright red areas seen under the low power in the van Gieson stained sections were, on more detailed examination, and especially when studied in hæmatoxylin preparations, found to be formed around small vessels, commonly an arteriole, sometimes with a vein



(Fig. 4). The walls of these vessels were hyaline and thickened, in many instances completely obliterating the lumen (Fig. 5).

In some cases thrombi in various stages of formation could be seen within the vessel. These thick-walled or obliterated vessels formed the centres of irregular areas composed of swollen glial fibres, necrotic granular tissue, and infiltrating round and fusiform cells with varying extent of hæmorrhage.

Around some small vessels groups of epithelioid cells were seen and, rarely, examples of the dissecting aneurysm in which the intravascular lymph space between the media and adventitia was filled with blood.

3. *Swelling and Necrosis of Tissue.*—In many sections, especially those near to the chief site of the hæmorrhage, the preponderating change was due to the exudation of fluid which had coagulated into homogeneous glistening masses.

Such masses were often found within the grey matter, entirely replacing the nervous tissue. This œdematous soaking led to swelling of the glia, revealed by thickened fibres compressed into fibrous-looking bands.

In Fig. 1 a large band is seen in the form of an arch immediately under the dorsal aspect of the grey matter, the white matter of the dorsal column thus enclosed was found in various stages of necrotic softening and destruction.

4. *Condition of the Nerve Elements.*—Whilst at the chief sites of disease both the cornual cells and nerve fibres exhibited all degrees of degeneration, these changes were almost entirely limited to the immediate region of vascular disturbance; beyond these zones, nerve fibres and nerve cells, studied by the van Gieson, Ströbe, and Nissl methods, were normal in appearance.

The substance of the cord and meninges was carefully examined for micro-organisms, but none found. The brain above the medulla was normal.

*Remarks.*—This is a noteworthy example of the class named "Acute Disseminated Myelitis":—vague toxic symptoms followed by profuse hæmorrhagic myelitis distributed over the whole of the spinal cord, extending into the medulla, and causing death within five days.

Similar cases are well known, usually following definite infection; they rarely, however, present so fulminating an onset or so rapid a course. Sir Thomas Barlow (1), many years ago,

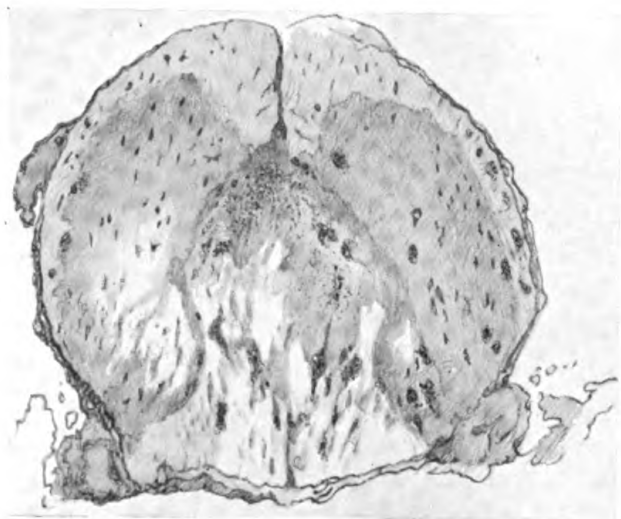


FIG. 1.



FIG. 2.



FIG. 4.

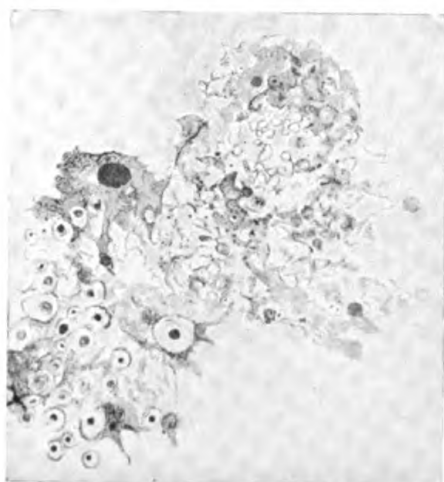


FIG. 3.

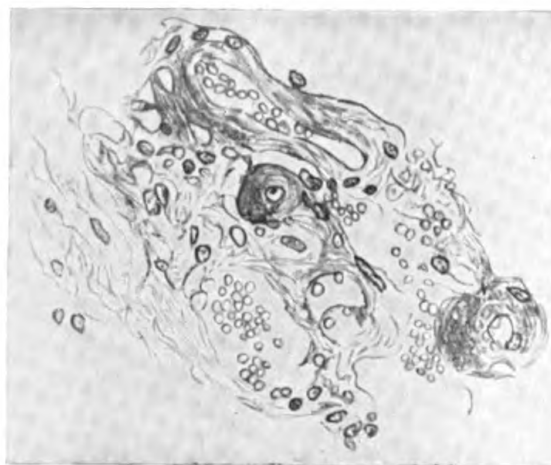


FIG. 5.



described a disseminated myelitis which terminated on the eleventh day of an attack of measles and on the third day from the first spinal symptoms.

Schiff's (2) patient, with typhoid, died in eighteen hours from the first appearance of palsy. In both these cases there was considerable hæmorrhage into the spinal cord, but this is not an essential feature of the disease. In most of the recorded fatal cases the duration of the illness was considerably over a week. Acute myelitis has for some years been the object of much pathological debate; we now only record the findings in a case carefully examined by modern methods.

It seems probable that most cases of transverse myelitis are rather examples of primary softening (*Myelomalacia*) due to impaired arterial supply, and, in the case of syphilitic disease, occurring early after the original infection. Williamson (3) has shown that the thrombosis of blood-vessels found by him was the primary cause of the softening and hæmorrhage, because thrombosed vessels were found far away from the hæmorrhage, many of the thrombi were clearly of older date than the hæmorrhage, and marked vascular change sufficient to account for the thrombosis was present; the known limitation of the inflammatory change to the distribution of one spinal artery, such as occurs in acute infantile palsy, is, as Batten (4) has recently pointed out, in favour of a primary thrombotic origin. Findings of this kind are valuable evidence, and are tests which should be applied to the interpretation of the microscopical appearances, for histologically the changes in simple and inflammatory softening *may* be identical; "the evidence of the initial thrombotic lesion may disappear in the inflammatory change it excites" (Sir William Gower's "Manual of Nervous Diseases").

Bruns (5) considers that etiological considerations can alone furnish a distinction between these different types of pathological changes; it is especially in the cases of acute disseminated myelitis that the evidence of bacterial or toxic invasion is clear, that the changes found resemble those produced by experimental inoculation (Marinesco), and that inflammatory action with necrotic softening follows the vascular distribution.

Mager (6) lays stress on the observed changes in the vessel wall, regarding these as an essential and primary expression of the activity of the virus, according to the nature and intensity

of which hæmorrhage, round-celled infiltration, or simple parenchymatous degeneration may result. Mager, however, considered the changes in the vessel walls as essentially of the same order whether they exhibit the characteristic thickening of the intima of syphilis or not, but of the seven cases described by him the changes in the intima were found only in the cords of those patients who had previously been affected with lues. We consider the changes in the vessel walls so clearly described by Williamson as occurring in syphilis are not comparable with those now described by us and frequently met with.

Singer's (7) cases also were examples of transverse myelitis in syphilitic subjects, and his conclusion that acute myelitis is pathologically a myelomalacia resulting from thrombosis in diseased arteries should refer specifically to that condition (acute transverse myelitis).

Against Mager's view of the primary nature of the vessel change such as we have described in our case, is the fact that these changes are not found in all cases; they were absent, for example, in Purves Stewart's case recorded in this Journal (1903), and which pathologically was a disseminated myelitis; in our case they were only marked in the chief sites of the disease, being absent in other regions of the cord where vascular dilatation and perivascular infiltration were present. It is noteworthy that they were found only in the smaller vessels, and we regard them as due in the first place to the intensity of the toxic virus, and secondly to the necrosing action of the inflammatory oedema. We have seen similar changes in the vessels of a spinal cord destroyed by the usual fracture dislocation of vertebræ and which ended fatally in four days. In conclusion, we think it desirable that further pathological reports should be published on cases of acute disseminated myelitis and encephalitis, which diseases at present we regard as being different in pathogenesis to acute transverse myelitis.

#### DESCRIPTION OF FIGURES.

FIG. 1.—Lumbar region stained by van Gieson's method, showing hæmorrhages and oedematous tissue in the dorsal columns, surrounded by a band of swollen glia, and dislocating the anterior cornua outwards and backwards.

FIG. 2.—Dorsal region stained by van Gieson's method, showing hæmor-

rhages in the distribution of the anterior spinal artery, and also in a slighter degree from the coronal vessels.

FIG. 3.—Necrotic masses surrounding a blood-vessel at the junction of the grey and white matter (Leitz, 1-12th hæmatoxylin).

FIG. 4.—Inflammatory focus around vessels in the posterior column (Leitz, 1-8th hæmatoxylin).

FIG. 5.—Vessels from a hæmorrhagic focus undergoing hyaline degeneration, and showing round celled infiltration, less marked here than in other preparation (Leitz, 1-8th hæmatoxylin).

Drawings made with the Edinger apparatus and Leitz' drawing ocular.

#### REFERENCES.

1. Barlow. *Trans. of Roy. Med. Chir. Soc.*, p. 77, 1887.
2. Schiff. *Archiv. für klin. Med.*, p. 175, vol. lxvii.
3. Williamson. "Syphilitic Diseases of the Spinal Cord," 1899, p. 62.
4. Batten. *Brain*, p. 376, 1904.
5. Bruns. "Twentieth Century Medical Practice."
6. Mager. Ref. in *Neurol. Centralbl.*, p. 455, 1900.
7. Singer. *Brain*, p. 332 (a), 1902.

*Note.*—A grant towards the expenses of work in Neuropathology is made to Dr Warrington by the Scientific Grants Committee of the British Medical Association.

## A CASE OF HÆMORRHAGE INTO THE BRAIN AND SPINAL CORD FROM OBLITERATIVE ARTERIAL DISEASE.

By W. B. WARRINGTON, M.D., F.R.C.P., and JOHN OWEN, M.B.

(From the David Lewis Northern Hospital and Thompson-Yates Laboratories, University of Liverpool.)

**SUMMARY.**—Girl, æt. 25. Apoplexy. Marked unilateral optic neuritis, endarteritis proliferans, rupture of aneurysm, hæmatoma of one optic nerve sheath, foci of hæmorrhagic softening around obliterated blood-vessels in the spinal cord.

A well-nourished young woman, aged 25, was sent into hospital on Aug. 15, 1905, by Dr Stack, of Wavertree. She was moaning and crying, and complained bitterly of pain in the head. When not calling out, she lay in a partially stuporose state.

The history of her illness was that eleven days ago, sitting at the tea-table, apparently in good health, she cut her finger. Convulsive movements of the left side of the body began, and continued for an hour, when she passed into a semi-

comatose condition, which continued for several days. At the end of that time she recovered sufficiently to recognise her friends and talk to them. She complained of much pain in the back and head, and from time to time relapsed into the semi-stuporose condition in which she was found on admission to hospital.

The girl was engaged as a housemaid, but recently had been doing the work of a general servant, and is said to have been overworked. No history of previous illnesses could be obtained, though lately the patient had complained of sore throat, and the menses had been too frequent.

She came of respectable and healthy parents, had several brothers and sisters in good health, with the exception of a grown-up sister, who for two years had suffered from fits.

Special enquiry was made as to any evidence of hereditary or acquired syphilis. The former could probably be excluded, and there was no evidence of the latter, though a frank statement from the mother suggested that the risk of infection could not absolutely be excluded.

*State on Admission.*—Examination of the body and viscera showed no signs of syphilis or tuberculosis. The heart was normal in size ; a faint systolic bruit heard at the apex. Urine and fæces were voided involuntarily, though from time to time it was necessary to pass a catheter. The urine was alkaline and contained a large amount of pus, but no casts. After separating the pus, only a small amount of albumen remained in the filtrate.

The pulse-rate varied from 90 to 120 per minute, and the temperature was slightly subnormal. The ears, throat, and nose were normal.

*Nervous System.*—There was marked left hemiplegia, including the face. The loss of power in the left limbs seemed very complete, but the patient was unable to aid us in our examination. We considered that there was also loss of sensation on this side.

The right upper limb and right side of the face moved fairly well, but the lower limb seemed paretic. Both plantar reflexes were of the extensor type, the response being more distinct from the left foot. The knee, ankle, abdominal, and epigastric reflexes could not be obtained on either side. The wrist-jerk was, however, present on the right, not on the left side.

*Eyes.*—The right pupil was larger than the left ; both reacted to light and accommodation. There appeared to be some paralysis of the upward and outward movement, but it is impossible to make a precise statement on this point. Particular interest attaches to the result of ophthalmoscopic examination.

Mr A. Nimmo Walker, of St Paul's Eye Hospital, noted as follows:—

“The right disc was extremely swollen, being best seen with a + 5 lens, and appeared like a reddish tumour. Its lower border was not to be defined, but superiorly it formed a prominent ridge overlapping the superior retinal vein. This was the only vessel to be seen, and around it was the only comparatively clear portion of the fundus. The vein was swollen, and ran vertically downward, apparently behind the swelling representing the disc. Its companion artery was quite invisible, but on each side of the vein were small dots of black pigment.

“The picture was one of intense congestion and exudation occurring in an eye which had previously suffered from chorio-retinitis.

“Owing to the restless delirium of the patient and the haziness of the media, it was possible to obtain only fleeting glimpses of the fundus.”

The left eye showed slight distinct optic neuritis, with some swelling of the veins and smallness of arteries.

*Progress of the Case.*—The patient lived for twenty-seven days, the symptoms remaining stationary. The stupor deepened into coma, and the temperature rose for a week or so before death.

Lumbar puncture was performed three times. On the first occasion, six days after admission, a reddish fluid was obtained, which was sterile, and showed only red blood-cells with a few leucocytes. Four days later the fluid was yellowish, and a week later normal.

*Autopsy.*—The pyuria was found to be due to a slight cystitis, there was no disease of the kidneys, and the other thoracic and abdominal viscera were normal, with perhaps the exception of a slight mitral stenosis not of recent origin ; the cusps of the valve were quite smooth, a trifle thickened, and there were no visible vegetations.

*Brain.*—On removal of the brain a fair amount of yellowish-



red fluid was seen, and masses of clot along the base of the brain extended from the pons forward. On the right side the optic foramen was plugged with clot compressing the nerve, the left nerve passed freely into its foramen.

The vessels at the base exhibited marked pathological change. The whole system was dissected away from the brain, and pieces from the vertebral, basilar, circle of Willis, and cerebral arteries examined with the microscope. To the naked eye they appeared as yellowish thickened tubes, and on cross section the diminution in the size of the lumen was clearly seen.

This thickening was of unequal distribution, adjacent parts of a vessel being either much diseased or practically free; both sides suffered equally.

On microscopic examination the thickened vessels showed well-marked endarteritis proliferans. In some sections the endarteritis produced a uniform thickening; in many others there were nodes formed by local overgrowth of the intima. Stained by acid orcein the characteristic reduplication of the elastic lamina was well seen. No tubercle bacilli were found after the appropriate staining. The change seemed limited to the larger vessels, the smaller cortical branches appeared normal.

Shortly after its origin the middle cerebral artery divides into four branches; the most posterior is the parieto-temporal, which lies under the temporo-sphenoidal lobe. Arising from the wall of this vessel at its origin from the common trunk on the right side a small sac the size of a pea was seen. A section was obtained through the sac and through the two more anterior branches of the middle cerebral; it was then seen that whereas the two latter vessels showed marked nodose proliferation of the intima, the wall of the sac was in places extremely thin, being represented only by a band of connective tissue. The rupture of this sac was no doubt the cause of the hæmorrhage, and the arrest of the circulation the cause of the reddish necrotic condition of the neighbouring parietal and temporal convolutions. Examination showed that the softening was not, however, confined to these areas, but affected the island of Reil and part of the ascending frontal convolution. The basal ganglia and internal capsule appeared normal to naked-eye examination.

*Spinal Cord.*—After formalin hardening, on cutting across the cord, brownish-red irregularly shaped spots could be seen in

the grey matter at various levels. Sections were stained by the Van Gieson, Ströbe, and acid orcein and hæmatoxylin methods. There was found considerable dilatation of blood-vessels throughout the whole cord, but no perivasular infiltration or distension of perivasular sheaths. Small hæmorrhages almost entirely confined to the grey matter occupied areas in the anterior and posterior cornua, and less frequently to one side of the central canal. The hæmorrhage had destroyed and torn the tissue in which it occurred without producing much change in the surrounding neighbourhood. There was no evidence of inflammatory reaction. Within many of these areas of hæmorrhage were seen small vessels, the lumina of which were almost



FIG. 1.

Small obliterated artery surrounded by an area of hæmorrhagic softening. (Leitz, 1-12th hæmatoxylin.)

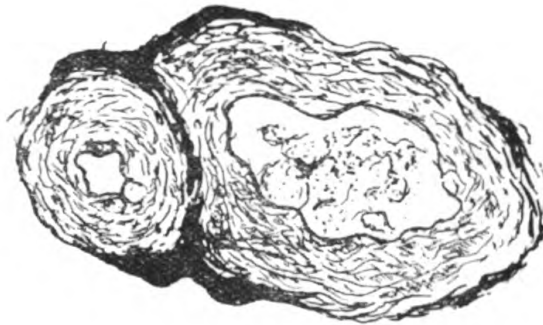


FIG. 2.

Thickened arteries in the spinal grey substance. (Leitz, 1-12th.)

*Drawings made with Leitz' drawing ocular.*

completely obliterated by thickening of their walls (Fig. 1). Many of the larger vessels in the grey matter also displayed a similar thickening of the walls (Fig. 2), but in no instance was any proliferation of the intima seen such as occurred in the cerebral vessels. The nerve elements showed little change, but Marchi and Nissl's method were not used. The glia was somewhat thickened in the neighbourhood of the dilated vessels.

*Commentary.*—Cerebral hæmorrhage was diagnosed during life from the presence of blood in the cerebro-spinal fluid; it was due to the rupture of a small aneurysm forming on an artery weakened by the advanced endarteritis proliferans so characteristic of syphilis. A similar change may be produced by other infections, notably tubercle, of which a beautiful

example is figured in Ziegler's Pathology. Ziegler says that the nodose form of endarteritis is by no means always due to syphilis. We refrain from expressing an opinion as to the causation in our case, though perhaps the chorio-retinitis mentioned by Mr Walker and the history may leave a suspicion in the mind.

Optic neuritis in cerebral hæmorrhage, apart from tumour, is a rare event. In a discussion at the Ophthalmological Society in 1881, Sir William Gowers (1) related the case of a child æt. 10, the subject of hereditary syphilis, with marked double optic neuritis; endarteritis with hæmorrhage into the optic nerve sheath was found post-mortem. A valuable paper by Remak (2) in 1886 showed that in cases of cerebral hæmorrhage with optic neuritis the bleeding extended into the optic nerve sheath, and that the neuritis was always more marked on the side of the lesion. The explanation given was compression of the central vessel by clot and interruption of the flow of lymph; this was doubtless the cause in our case, though we have to regret that this was not definitely confirmed by microscopical examination.

The appearance of the fundus was remarkable for the intense red congestion and is one we have not seen before.

The condition, then, of hæmatoma of the optic sheath and its frequent unilateral limitation is one of some clinical importance.

The mild papillitis on the opposite side cannot be explained in a similar way: it may have been due to an infective blood state or to local alteration in the retinal vessels.

The evidence in favour of syphilis as a blood infection producing optic neuritis is very slight. Emile Berger (3) alludes to such cases and quotes Horstmann, who collected eight cases; these, however, showed endarteritis.

In the spinal cord the findings may be contrasted with those present in the case of myelitis. The van Gieson specimens show a superficial resemblance in the distended vessels, areas of hæmorrhage, and the bright red of the swollen glia, but detailed examination suggests that here the hæmorrhages are due to obliteration of small blood-vessels, for these were found in many of the hæmorrhagic foci; the surrounding tissue had suffered a local neurosis and showed no signs of inflammatory reaction; thickened blood-vessels were found elsewhere in non-affected

areas and in the brain; in the vertebral arteries, from which the anterior spinal arises there was pronounced disease.

Batten (4) quotes the old experiment of Prevost and Cotard, as showing that hæmorrhage may be due to emboli in small arteries; and Williamson (5) mentions the observations of Lamy, who, on injecting inert powder, found foci of hæmorrhagic softening in the grey substance, the region of distribution of end-arteries.

#### REFERENCES.

1. Gowers. *Trans. Oph. Soc.*, vol. i., 1881
2. Remak. *Berlin klin. Woch.*, pp. 828 and 848, 1886.
3. Emile Berger. "Les Maladies des Yeux," 1892.
4. Batten. *Brain*, p. 376, 1904.
5. Williamson. "On the Relation of Diseases of the Spinal Cord to the Distribution and Lesions of the Spinal Blood-vessels," 1895.

*Note.*—A grant towards the expenses of work in Neuropathology is made to Dr Warrington by the Scientific Grants Committee of the British Medical Association.

---

## Abstracts

### ANATOMY.

**NOTE ON THE TAENIA PONTIS.** Sir VICTOR HORSLEY, *Brain*, (208) April 1906, p. 28.

THE author, in a number of photographs of the brain of the camel, hippopotamus, and man, shows the position of a bundle of fibres running antero-laterally along the upper border of the pons, to which Heule applied the term *taenia pontis*. It was regarded as arising in the cerebellum and ending in the pons varolii, but the exact origin and termination of its fibres was not known.

In this note, two cases of congenital defective cerebellum in the human subject are described, in which this bundle was traced from a "mass of embryonic grey tissue continuous with that of the interpeduncular ganglion" of one side in which it seemed to arise, to the region of the dentate nucleus of the other side. To determine the direction of its fibres, the author divided the *taenia pontis* in a monkey and traced the resulting degeneration by the Marchi method. He found that all its fibres between the lesion and the cerebellum were degenerated, their direction being therefore cerebello-petal, like the other ponto-cerebellar systems in

parallel position. The degenerated fibres were followed to the dentate nucleus of the opposite side, in which they appeared to end, a few passing inwards to terminate in the roof nuclei.

SUTHERLAND SIMPSON.

**A NOTE ON THE TOPOGRAPHICAL ANATOMY OF THE  
(209) CAPUT GYRI HIPPOCAMPI.** JOHNSON SYMINGTON, *Journ.  
of Anat. and Physiol.*, April 1906, p. 244.

THIS part of the gyrus hippocampi is of considerable morphological and physiological importance, since it corresponds to the pyriform lobe of mammals and is the main cortical centre for smell. Its boundaries and certain markings on its surface have been investigated with great care by Prof. G. Retzius, who has described a fissure on its surface which he terms the *sulcus rhinencephalous inferior*.

During the examination of a series of adult human brains hardened *in situ* by the injection of formol, Symington invariably found a groove in the position of the fissure described by Retzius, and by making dissections of the brain *in situ*, this groove was seen to be occupied by the anterior part of the free edge of the tentorium cerebelli, a little behind the anterior clinoid process and external to the third nerve. These relations are most readily shown by making a median section of the head and then dividing on each side the upper part of the crus cerebri and removing the portion of the brain below this cut. By this means the relation of the free edge of the tentorium cerebelli to the brain is easily ascertained. On such preparations it will be seen that this edge passes forwards and downwards from just behind the splenium of the corpus callosum across the isthmus, uniting the callosal and hippocampal gyri, and occasionally indenting it. It then passes into the depression between the upper border of the lateral aspect of the pons varolii and the crus cerebri. In front of this it again comes into close relation with the cerebral cortex, and on the caput gyri hippocampi corresponds to a distinct longitudinal depression. The lower part of the caput lies against the dura mater in the middle fossa of the base of the skull, where the dura mater forms the outer wall of the cavernous sinus. The upper part bulges upwards and inwards above the edge of the tentorium and presents an uneven surface, being marked by the gyrus lunaris and gyrus ambiens of Retzius and continued backwards to form the uncus. This depression cannot be regarded as a true cerebral fissure, but it is of interest as an illustration of the close relation of the basal portion of the cerebral cortex to the cranial wall and dura mater.

AUTHOR'S ABSTRACT.

**THE RADIX MESENCEPHALICA TRIGEMINA AND THE**  
(210) **GANGLION ISTHMI** J. B. JOHNSTON, *Anatomischer Anzeiger*,  
1905, S. 364.

THE mesencephalic root of the trigeminus of the vertebrates has been generally regarded as a motor structure, and it has been assumed that its fibres spring from the large unipolar cells which lie along its course. But neither of these assumptions has been yet proved.

The nucleus magnocellularis tecti of the fish brain has been regarded as the analogue of the nucleus of the upper root of the trigeminus. The author's investigations on it were made on the brain of *Scyllium* by Weigert's medullary sheath stain. He finds that the fibres which form the mesencephalic tract always enter the brain with the sensory root, quite separate from the motor fibres. During their course forward they pass beneath the decussation of the trochlear nerve and the decussatio veli into the midbrain, where they end chiefly in the fibre layer of the tectum: it is doubtful if any of them have any connection with the nucleus magnocellularis. In *Acipenser*, too, he has now recognised the existence of an ascending trigeminal root, which also ends in the fibre layer of the tectum and in the torus semicircularis. This had been previously described by Goronowitsch, but was denied by Johnston in his monograph on that brain. The tectum is therefore a primary centre of somatic sensation; this is of interest in view of the fact that the tectum is regarded as the oldest sensory centre.

The second portion of the paper refers to the nature and significance of the nucleus, which has been described by Edinger in all classes of vertebrates, except the mammals, by the name ganglion isthmi. It was earlier known to Mayser as the Rinden-knoten, and he recognised that the secondary vagal tract ended in it. It is now known that it is the centre in which the fasciculus communis, which is the secondary tract of visceral components from the seventh, ninth, and tenth sensory nuclei terminates, and it must, consequently, be regarded as the secondary visceral nucleus.

Finally, attention is drawn to the relation of the cerebellum to the valvula cerebelli. The latter is met with only in the teleosts and ganoids, in which the gustatory system is well developed. It seems probable that the cerebellum proper belongs to the somatic sensory system, while the valvula cerebelli is part of the visceral sensory apparatus especially developed in relation to the olfactory and gustatory senses.

GORDON HOLMES.

**CONTRIBUTION TO THE STUDY OF LOCALISATION IN THE**  
**(211) NUCLEI OF THE CRANIAL AND SPINAL NERVES IN**  
**MAN AND IN THE DOG.** (Nouvelle contribution à l'étude  
 des localisations dans les noyaux des nerfs craniens et  
 rachidiens chez l'homme et chez le chien.) C. PARHON and  
 G. NADEJDE, *Journ. de Neurol.*, April 1906, p. 129.

IN a case of cancer of the tongue which ended fatally in two years, and in which at the time of death this organ was almost completely destroyed and many of the muscles of the sub-hyoid region also involved, including the sterno-cleido-mastoid, the authors examined the spinal cord, medulla oblongata, and pons by Nissl's method, with a view to determine the positions of the group of cells affected.

The central group of cells in the anterior horn of the first and second cervical segments they believe to be the nucleus of the sterno-cleido-mastoid muscle. This group does not extend into the third segment. All the cells of the upper part of the hypoglossal nucleus were atrophied except the anterior group, which showed signs of recent change, and as the genio-hyoid muscle was known to have become invaded only towards the end of the disease, this group they associate with the nerve supply of that muscle.

In the nucleus of the seventh, the group, which they term the "second ventral group," is the centre for the posterior belly of digastric, stylo-hyoid and stylo-glossus muscles, and from a difference on the two sides they believe the most external cells of this group to belong to the last named muscle. The anterior belly of the digastric and the mylo-hyoid they find to be represented in the chief motor nucleus of the fifth, these being supplied by the mylo-hyoid branch of the fifth nerve.

These findings in man they have confirmed experimentally in the dog, and in addition in this animal they have located the position of the centres for the innervation of the temporal, masseter, and pterygoid muscles in the motor nucleus of the fifth.

SUTHERLAND SIMPSON.

**RESEARCHES ON THE MINUTE STRUCTURE OF THE NERVE**  
**(212) CELL IN VERTEBRATES.** (Ulteriori ricerche sulla intima  
 Struttura delle Cellule nervose nei Vertebrati.) ROSSI, *Le*  
*Nervaze*, April 1, 1906.

THIS paper is a continuation of the author's previous work on the reticulum in the nerve cell. Using Cajal's and Donaggio's methods he can demonstrate, in the spinal nerve cells of vertebrates and in

man, two networks, one at the periphery composed of coarse fibrils intimately connected with each other and preserving in the cell body the direction of the protoplasmic processes from which they come; another situated nearer the centre of the cell composed of very fine fibrils united to form a true network and connected with the peripheral one. The author's peripheral network is not identical with the external reticulum of Golgi.

By using the chloride of gold method he can demonstrate an endocellular reticular structure resembling that shown by Golgi with his nitrate of silver method.

This structure is delicate and filamentous, tortuous, and its elements are of uniform calibre. The filaments anastomose and the reticulum rather resembles that observed by Apathy in the leech and by Gemelli in the worm.

The author has not yet come to any conclusion as to the interpretation to be put upon this appearance. DAVID ORR.

**A STUDY OF THE MINUTE STRUCTURE OF THE OLFACTORY  
(213) BULB AND CORNU AMMONIS, AS REVEALED BY THE  
PSEUDO-VITAL METHOD. With remarks on the plan of  
Nervous Structure of Vertebrates in general. JOHN TURNER,  
*Brain*, 1906, p. 57.**

THE author's method is described by which a *differential* staining of the central nervous system is obtained, and extra-cellular neurofibrils shown. The method is not applicable to cord or spinal ganglia.

The brain is cut into pieces about 3 mm. thick, and laid on the flat bottom of an inverted glass capsule, which is placed in a larger receptacle, surrounded by distilled water, to which has been added eight drops of formalin. The whole is covered by a glass plate, and subjected to a temperature of 24°-25° C. for 30 hours. The pieces are then placed in a stoppered bottle in 15 c.c. of a 1% solution of patent methylene blue (Grübler) containing 12 minims of lactic acid to each 100 c.c. One or two cubic centimetres of dioxygen (3% solution of hydrogen peroxide) are added to the stain, and the bottle kept for five days at the before-mentioned temperature. The pieces are then rinsed, and placed in a fresh 10% solution of molybdate of ammonium, acidulated with two drops H.Cl., and left for 12 hours; washed under running water all day; dehydrated in alcohol, passed through chloroform; imbedded in paraffin, and cut.

The appearances presented by the olfactory bulb of animals when stained in this manner are then described, and the similarity



of its microscopical features to those of the cerebellum pointed out, and it is suggested that these organs are homologous.

The structure of the cornu ammonis and fascia dentata are next described, and an attempt made to homologise their layers with those of the pallium. It is suggested that the stratum oriens corresponds to the fusiform or spindle cell layer; the stratum pyramidalum and s. radiatum to the large pyramidal layer; the s. lacunosum (and s. granulosum of dentate fascia) to the second or small pyramidal layer.

The uniform structure of all grey matter, cortical or central, is pointed out, and the great importance in the evolution of the mammal's brain of the inter-cellular plexus of neuro-fibrils—this plexus assuming, as one passes from lower to higher forms, an increasing richness and delicacy.

The features in the development and finer structure of the vertebrate nervous system which favour the assumption of *continuity* in the neuro-fibrils are briefly discussed, and schemes both of the reflex arc apparatus, and of the nervous system in general, on the lines of continuity of neuro-fibrils, are given.

AUTHOR'S ABSTRACT.

#### THE CRANIAL NERVE COMPONENTS OF PETROMYZON.

(214) J. B. JOHNSTON, *Morpholog. Jahrbuch.*, 1905, Bd. 34, S. 149.

THIS paper deals with the complicated subject of the arrangement and functions of the cranial nerves of the fishes. Petromyzon was selected for this study, as it was expected that the condition in the cyclostomes would throw light on the structure of the primitive vertebrate head. As only the constitution and distribution of the peripheral portions of the nerves are here considered, a full abstract of the paper is scarcely possible, but attention may be drawn to some interesting points in the conclusions.

Any nerve trunk may be made up of fibres belonging to two or more different systems. There are five systems represented in the cranial nerves—general cutaneous, special cutaneous (which supply the pit organs), visceral sensory, visceral motor, and somatic motor. The author has succeeded in analysing each nerve into its separate components. His conclusions are:—

In the arrangement of its cranial nerve components Petromyzon agrees with other fishes, but has some marked primitive characters.

The facialis contains a general cutaneous component for a portion of the hyoid segment; these fibres are not represented in the higher fishes. General cutaneous components are also found in the glossopharyngeus and vagus, and the dorsal spinal nerves

contribute similar components to the epibranchial trunk of the vagus.

The arrangement of the pit organs and of the rami innervating them is in general the same as in other fishes, except that those in the branchial region are innervated by special cutaneous components from the glossopharyngeus and epibranchial trunk.

The visceral sensory component is very small in the facialis, but large in the glossopharyngeus and vagus.

Large taste organs on the internal surface of the pharynx, between each pair of gill slits, are innervated by visceral cutaneous fibres from the glossopharyngeus and from each branchial nerve.

The first two ventral nerves innervate the first three post-otic myotomes. Only the abducens and the first spinal nerve are absent. A dorsal nerve is present in each segment beginning with the nervus ophthalmicus profundus.

A sympathetic trunk and ganglia are present in the head.

GORDON HOLMES.

**THE CRANIAL AND SPINAL GANGLIA AND THE VISCERO-**  
**(215) MOTOR ROOTS IN AMPHIOXUS.** J. B. JOHNSTON, *Bio-*  
*logical Bulletin*, Vol. i., No. 2, July 1905.

ROHDE and Hatschek described collections of nerve cells on the dorsal roots of amphioxus, which they regarded as analogues of the spinal ganglia of the higher vertebrates.

Retzius, Dogiel, and Heymans and van Stricht, however, failed to find them, and came to the conclusion that the dorsal roots arise from intraspinal cells.

The author, by the aid of the intra-vitam methylene blue injections and by Golgi's method, has been able to demonstrate four different classes of cells from which dorsal root fibres take origin: (1) Bipolar cells, one process of which enters the dorsal root while the other extends into the opposite half of the cord. (2) Similar bipolar cells, but their central processes remain on the same side of the cord. (3) Bipolar cells in the root or trunk of the nerve, which are probably identical with those described by Rohde and Hatschek. (4) Irregular cells, provided with a thick process which breaks up near the surface of the cord and sends a fine fibre into the dorsal root. These are probably the fibres which supply the viscera. Thus only part of the spinal ganglia are intraspinal.

The greater portion of the dorsal root fibres end in or about the segment at which they enter; the rest form three tracts in the dorsal part of the cord, a diffuse mesial tract of fine fibres, a diffuse lateral tract of coarser fibres, and a compact dorsal tract

of coarse fibres. The latter is the earliest representative of the dorsal columns of the mammalian cord, and it is the first tract to appear as a definite bundle in the vertebrate nervous system.

The central nervous system of amphioxus resembles in many particulars that of the lower fishes. GORDON HOLMES.

**APPLICATION DE LA MÉTHODE DE RAMON Y CAJAL (IM-  
(216) PRÉGNATION À L'ARGENT) À L'ANATOMIE PATHO-  
LOGIQUE DU CYLINDRAXE.** THOMAS, *Revue Neurol.*, March  
31, 1906, p. 249.

THE author strongly recommends the above method for the study of the morbid changes in axis-cylinders. The tissues employed were taken from cases in which there was degeneration of the pyramidal tracts. The axis-cylinders were greatly reduced in number, a few only remaining stained black, with or without a myelin sheath. Some were distorted and swollen, others much atrophied. Moniliform swellings were common.

DAVID ORR.

**ON THE ESTIMATION OF SKULL CAPACITY ON THE CADAVER.**  
(217) (Ueber die Bestimmung der Schädelkapazität an der Leiche.)  
M. REICHARDT (of Würzburg), *Allg. Ztschr. f. Psych.*, Bd. 62,  
H. 5, 6.

UNLESS the disproportion between the brain and the cavity of the skull is well marked it is impossible, according to the author, to say definitely whether the brain is of its normal size, or atrophic or swollen. The weight itself of the brain is insufficient; the important question is, whether the brain in relation to the cavity is too heavy or too light; Reichardt gives in detail his method for the estimation of skull capacity. C. MACFIE CAMPBELL.

## PHYSIOLOGY.

**THE LOCALISATION OF THE HIGHER PSYCHIC FUNCTIONS,  
(218) WITH SPECIAL REFERENCE TO THE PREFRONTAL  
LOBE.** By CHARLES K. MILLS and T. H. WEISENBURG  
(Philadelphia), *Journ. Am. Med. Ass.*, Feb. 1906.

AFTER giving a short history of cerebral localisation from the first enunciation of the theories of Gall to the definite localisation of

the motor and sensory functions in the middle and posterior part of the cerebral cortex, the authors go on to point out that Flechsig's embryologic researches (1893) demonstrated the existence of cerebral association areas concerned with intermediate and higher psychic functions. Since then the subject has evolved in two directions: (1) in the direction of a more exact delimitation of areas concerned with primary functions; (2) in the recognition of sub-divisions of Flechsig's posterior association area, the concrete memory field, especially as regards the visual and auditory sub-divisions. The authors then give their views as to the higher psychic functions, and state that they believe that the highest mental faculties or functions have their material representation in the prefrontal lobes of the brain, and especially in the left prefrontal lobe. After pointing out the mistake made by Gall and the old phrenologists that they did not recognise the essential difference between a fundamental faculty or function and certain cerebral side attributes or epiphenomena, they state that whilst the ability to attend, to will, to judge, to compare, to reason, and to exercise the imagination are fundamental psychic functions, the exhibition by an individual of pride, of vanity, of friendship, of combativeness, of piety, etc., is not due to the operation of any single mental faculty. The fundamental psychic functions are represented by special centres associated by special tracts, whilst the latter so-called faculties result from the action of one or several or many of the regions of the brain. The authors put forward some of the evidence on which their opinions are based.

A careful study of the brains of notable men has shown the high development of the prefrontal region, whilst if these are put alongside of brains of low individual or racial development—as criminals, imbeciles, negroes, etc.—the difference is marked in the prefrontal region.

The embryologic researches of Flechsig and the histological investigations of Campbell show the absence of projection cells and fibres in this portion of the brain. It is the very last pallium to appear in the progress of phylogenesis. The authors then point out that gross and microscopical examination of the brains of general paretics support in some degree the thesis that the prefrontal portion of the brain is the seat of its highest functions. They discuss the question as to whether the prefrontal region is divided into sub-areas or centres, if it be granted that it is the seat of the highest intellectual functions. After stating how difficult it must be separating the highest psychic functions from each other, as they are so interblended in expression, they state that a clue may be found to their relative position on the higher psychic region by a consideration of the positions and relations of the centres of known functions contiguous to the prefrontal lobe.

As Broca's centre is situated just anterior to the centres of articulation, enunciation, and phonation, the sub-divisions of the so-called face areas, so speech, being the chief instrument employed in reasoning, one would expect the psychic centres of ratiocination to be topographically closely related in position to the centre of Broca. In the same way, writing, administering to exactness in thinking, the centres of comparison and judgment may have their highest development contiguous to the graphic centre. Again, whilst the motor centres for the limbs and body are in the pre-central convolution, the centres for movement of the head and eyes, especially the latter, are thrust towards and into the pre-frontal lobe. Attention and volition have their most marked physical expression through vision and the position of the head, and if there are centres for attention and volition, their most probable situation is in parts contiguous to the centres for movements of the eyes and head.

The authors then cite the statistics of Phelps, Müller, Schuster, von Monakow, and others, and point out how they in many respects support the views here enunciated. Many fallacies, however, are apt to creep not only into the statistics of mental cases, but also in the recording of the facts, for many reasons which are discussed by the authors.

A case of their own observation is recorded in support of their views. A physician, seventy-one years of age, who till within a few months of his death carried on his profession, was seen about two months before death by Dr Mills when his symptoms were all mental, chiefly exhibited in defects of memory and judgment. He had no paralytic or sensory symptoms. Shortly afterwards he was noticed to have a shuffling gait, and later was unable to carry food to his mouth with any certainty. Then he developed muscular tremors in his hands and legs like those of paralysis agitans. He died comatose. His son, a physician, supplied observations after his death showing that the patient was affected in his higher psychic functions, in his judgment and powers of comparison, in his grasp of his work, in his disposition, etc.

At the necropsy the lesion was found to be a left prefrontal tumour, fleshy in colour, hard in substance, and well defined from the surrounding tissues. It extended as far back as the knee of the callosum and the caudatum and anterior extremity of the internal capsule, but was limited on the surface to the anterior part of the first, second, and third frontal convolutions. Two photographs show the extent of the lesion.

WILLIAM ELDER.

**AN OSMOTIC THEORY OF SLEEP.** (Théorie osmotique du (219) sommeil.) E. DEVAUX, *Arch. gén. de méd.* April 10, 1906, p. 903.

THE author supposes that the cells of the nervous system, parting in the course of their activity with various molecules of their composition, become more and more greedy for water, which they attract from the blood. This process continues until there is a steady flow of serum from the capillaries into the tissues. The blood in the capillaries thus becomes thick and viscous. The terminal arterioles become constricted. The flow through the capillaries becomes very slow. The consequence of this is twofold. Firstly, a copious nutriment is brought to the cells, which thus are able to restore their reserves. Secondly, the necessarily diminished supply of oxygen reduces the sensibility of the cells, hence the condition—sleep.

The theory is supported by several arguments:—

1. If sleep is due to a flow of plasma from the vessels, the blood pressure should fall. This is what actually does occur.
2. This fall of pressure is certainly not due to a lowering of the peripheral resistance, for in that case the pulse-rate would rise, whereas it falls.
3. If a smaller quantity of blood circulates during sleep, we should expect to find a diminished secretion of urine. This is just what does occur.
4. If there is less fluid in the vessels during sleep, there must be more in the tissues. This seems to be indicated by the tendency to cedema, as expressed, for instance, in the saying, "His eyes are still heavy with sleep." The duration of imprints made upon the skin by the pressure of a definite weight for a definite time is longer when the trial is made during sleep.
5. During sleep, breathing is shallower and the flow of blood through the lungs is slowed. One would, therefore, naturally expect a diminished absorption of oxygen. As a matter of fact, the oxygen absorption is much increased—a fact which may be explained on the theory that the blood, deprived of so much of its serum, has become relatively very rich in corpuscles.
6. Lastly, Dastre and Loge have shown, and their observation has been confirmed by Langlois, that during chloroform narcosis, massive injections of artificial serum isotonic with the blood serum are not excreted by the kidneys or other organs of elimination, but accumulate in the tissues—a striking confirmation of the theory that during sleep there is a marked osmotic current from the blood towards the tissues.

W. B. DRUMMOND.

**THE CONDUCTING PATHS FOR PAINFUL AND THERMAL  
(220) IMPRESSIONS IN THE SPINAL CORD.** (*Les voies de la sensibilité douloureuse et calorifique dans la moelle.*) E. BERTHOLET, *Nevrage*, Vol. vii., F. 3, 1906, p. 283.

BERTHOLET begins his paper with an historic review of the previous work on this subject. All the authors quoted are practically unanimous in regarding the grey matter of the spinal cord as the conducting path for the sensations of pain and temperature. The second part of the paper is devoted to a detailed account of the experimental work which has led Bertholet to take a different view to that of former observers. The experiments were performed on cats and consisted in the production of various hemisections, three-quarter sections (destroying the grey matter on both sides), double hemisections, double quarter sections (destroying both lateral columns), and finally in the destruction of the central grey matter of the cord itself. The results obtained, briefly summarised, are as follows: single hemisection did not in any way interfere with the conduction of heat and pain sense from either side posterior to the level of the lesion; similarly three-quarter section caused no interruption of the passage of these sensations; on the other hand, double hemisection resulted in complete and permanent loss of heat and pain sense; double quarter section caused a temporary loss of painful sensation (this appears to have been a single experiment only); destruction of the central grey matter was followed by complete retention of pain sense on both sides. Bertholet, therefore, concludes that the grey matter is not the conducting path for painful or thermal impressions, but that it only serves as a bridge connecting the fibres of the posterior nerve roots to those of the lateral columns of the cord in which the conducting strands lie; he believes these to be contained in the tract of Gowers.

E. HEWAT FRASER.

**ON THE QUESTION OF EXHAUSTION FROM EXCESS OF  
(221) FUNCTION.** (*Beitrag zur Lehre vom Aufbrauch durch Hyperfunktion.*) LILIENSTEIN, *Munch. med. Wochenschrift*, April 17, 1906, p. 748.

THE object of the paper is to supply three instances of the incidence of disease falling on the part or parts of the body that had been in a state of functional over-activity.

A girl employed in a typefoundry suffered from plumbism, and the condition affected not so much the extensors of the wrist and fingers as the muscles supplied by the ulnar nerve. It was dis-

covered that the girl's occupation entailed a constant quick deviation of the wrist to the ulnar side when she was filing type.

Another girl employed as a telephonist developed neuritis in the right arm, seemingly attributable solely to continually holding a heavy receiver in the right hand. The adoption of another apparatus which did not necessitate the employment of the right hand was followed by a disappearance of the symptoms.

The third case is not quite so clear, weakness in an arm that had long since been fractured being conceivably attributable, not merely to occupation, but also to the consequences of previous syphilitic infection.

The author remarks on the importance of careful supervision of all cases of tabes practising Frenkel's exercises, inasmuch as some, so far from improving, actually deteriorate when undergoing the treatment. These are instances of exhaustion from excess of function.

S. A. K. WILSON.

## **PATHOLOGY.**

### **RESEARCHES ON REGENERATION IN PERIPHERAL NERVES.**

(222) (*Recherches sur la Régénérescence des Nerfs Périphériques.*)

MARINESCO and MINEA, *Rev. Neurol.*, avril 15, 1906, p. 301.

By the employment of Cajal's new staining method, the writers had previously come to admit the existence of so-called "autogenic" regeneration in peripheral nerves. They did not, however, deny that the nerve-cell exercises some trophic influence on the axon. The evidence in favour of autogenic regeneration they classified under two headings: (1) the presence of newly-formed fibres within the cell-bodies and in the protoplasmic processes of the proliferated neurilemma nuclei; (2) the existence of newly-formed fibres in the peripheral segment of nerves torn from the cord, together with the anterior roots and the intervertebral ganglia. The neurilemma nuclei play an important part in the mechanism of regeneration, not only in the peripheral but in the central segment.

Cajal, however, in a recent paper, defends the classic Wallerian theory of regeneration. He describes how in young animals young axis-cylinders grow out from the central segment, about the beginning of the second week. These young fibres, devoid of myelin sheaths, push across the cicatrix and penetrate into the peripheral segment, in spite of all obstacles which may be interposed. In immediate reunion the neurotisation of the peripheral segment occurs rapidly: where there are intervening obstacles, the process may require three or four months, or more. The fibres



growing from the central end are mostly continuations of the old fibres, and the increased number of new fibres is due to Y-shaped division of the old, chiefly in the substance of the cicatrix. Cajal denies the neuroblastic function of the neurilemma nuclei. He describes the growing extremity of the young fibre as possessing a "terminal ball," which, if it becomes impacted in one of the tissue-interstices, may become enormously swollen and enlarged. Cajal attributes three functions to the proliferated neurilemma nuclei: firstly, a phagocytic function removing the débris of the old degenerated fibres; secondly, that of producing guiding or orienting sheaths, whose function is to secrete and set free a chemiotaxic substance capable of acting on stray young axones; and, thirdly, the function of maintaining the nutrition of the young nerve fibres when they arrive.

Marinesco and Minea have pursued their study of the process of regeneration, chiefly by experiments on puppies and on young rabbits. In the main, their observations are corroborative of those of Cajal above described, but they draw somewhat different conclusions from the facts.

The existence of new interstitial nerve-fibres and their Y-shaped branchings, together with the frequency of the terminal balls, most of which are directed from the centre towards the periphery, have caused the authors to abandon their original views as to autogenic regeneration. They now believe that the new fibres in the peripheral segment are produced by outgrowth and branching of the fibres in the central stump. Nevertheless, they still uphold the immense importance of the part played by the neurilemma nuclei, and maintain that regeneration does not occur unless in the presence of proliferation of these nuclei, whose cell-colonies constitute, as it were, the advance-guard of the regenerative process. Even when the central nerve-cell is atrophied, by separation of the central segment from the nerve-cell, new nerve-fibres can still grow out from the central segment to the peripheral. To Cajal, however, belongs the credit of having demonstrated the existence of nervous plexuses, microscopically invisible, which permit the transmission of new fibres from the central into the peripheral segment. This plexus, they say, has escaped the notice of the upholders of the autogenic regeneration theory. If it be admitted that the new nerve-fibres are not born spontaneously within the protoplasm of the neurilemma-cells, Cajal's next point must, they believe, be conceded, that the fibres are attracted by the cells, thanks to some chemiotaxic substance elaborated by them. The authors, however, disagree with Cajal's view as to the phagocytic function of the neurilemma-cells, and believe that phagocytosis is performed by the ordinary phagocytes of the tissues.

PURVES STEWART.

**CEREBRAL SCLEROSIS.** CAMPBELL, *Brain*, Parts 3-4, 1905.  
(223)

THE author in this paper first discusses the origin of the several varieties of neuroglial elements in the brain, and the origin of the cells which have a phagocytic function in cerebral lesions. He then goes on to consider all the known forms of sclerosis which occur in the brain.

The first class includes the somewhat rare cases of what is called "tuberosc sclerosis." This condition is most frequently met with in idiots and imbeciles. In most cases there is a history of epilepsy, and it is almost always accompanied by the rare skin affection, adenoma sebaceum. In the kidneys, too, it is usual to find a number of small, hard, white tumours, which are thought to be endothelial neoplasms.

In the case described by the author the small tumours lay in the cortex cerebri and the adjacent white matter. They consisted of a dense network of proliferated neuroglial fibres in which a few pyramidal and fusiform nerve cells and old neuroglial nuclei were scattered. In the peripheral portions of these masses some peculiar tubular glands, lined with columnar cells, were found.

Other small white tumours, probably of endothelial origin, were seen in close connection with the venules on the ventricular surface of the basal ganglia.

The origin of these tumours must be attributed to some developmental anomaly of the central nervous system, which was the result of some process which became active about the seventh month of intrauterine life.

This assumption that these tumours are the outcome of some evolutionary aberration or disturbance, which results in structural hyperplasia and heterotopia of the grey matter, supports the view that cerebral sclerosis is almost always a secondary condition.

The term, evolutionary aberration, used in connection with this condition of tubular sclerosis, may be applied to several other conditions described by the author. These conditions include:—

1. Hypertrophy of the cerebrum, in which the brain is uniformly hypertrophied and indurated while the microscopic architecture is normal.

2. Hemisclerosis, in which there is a diminution in size, affecting one hemisphere alone, equally distributed, associated with sclerosis, but unaccompanied by disarrangement of the gyral or sulcal architecture. On the whole, the nerve cells preserved their columnar arrangement and their erect attitude, but the cortex was reduced in depth by quite two-fifths.

3. Lobar sclerosis and microgyria, in which various lobes may be affected on both sides of the brain.

4. Giant cell sclerosis. In this condition there is a heterotopic anomaly of the nervous system.

The next class includes those cases of cerebral arterio-sclerosis and colloid sclerosis which are produced by vascular changes.

Lastly, brief reference is made to several of the commoner scleroses, viz. those met with in general paralysis of the insane, in senility, in the cornu ammonis in epilepsy, after softenings, in microgyria, in cerebral syphilis, and in disseminated sclerosis.

R. G. Rows.

#### **HETEROTOPIA OF THE CEREBRAL CORTICAL SUBSTANCE.**

(224) (*Delle eterotopia delle sostanza corticale cerebrale.*) G. B.

PELLIZZI, *Ann. di Freniat.*, Vol. xv., Fasc. 4.

THIS is an enquiry into the relation which exists between idiocy and epilepsy, and heterotopia and other malformations of the cerebral cortex.

In the case quoted by the author there was a history of restlessness and irritability from birth, and at the age of four years definite epileptic attacks began; finally the patient died after a series of fits.

At the autopsy, the right hemisphere was larger than the left, especially in the posterior portion, and the convolutions were less distinct. The right ventricle was much dilated, especially the occipital horn. In the lower wall of this horn there was a conical depression which extended down to the pia mater over the occipital lobe.

The fusiform convolution was diminished in size, and all the surrounding convolutions were in a condition of microgyria by sub-division. The pia mater here was thickened and adherent. A vertical section through the brain exhibited the conditions of porencephaly, heterotopia of the grey matter, and microgyria by sub-division. Microscopically, the superficial cortex was very thin and often interrupted, and it was poor in nervous elements, which were always small and generally pyramidal in shape.

The heterotopic masses were surrounded by large bundles of myelinated fibres with normal axis-cylinders; small fibres were present within them. The arrangement of the nerve cells was quite irregular, and they were less numerous than normal.

Most of the cells here were globose, and only a few were pyramidal. Only a few giant cells were seen, and they were badly formed.

The presence of pyramidal cells in all the heterotopic masses proved their common cortical origin.

The neuroglia cells were more numerous than normal.

Heterotopia of the cerebral grey matter may affect the cortex or the basal ganglia. In the latter case there may be pedunculated small tumours on the floor of the lateral ventricles surrounded by a dense neuroglial layer which is in connection with the subependymal neuroglia by the peduncle. This form is met with chiefly in the brains of idiots, and is associated with disseminated hypertrophic sclerosis.

It may be present also in the form of small tumours under the ependyma, and surrounded by it. This condition is found in adults and old people, and sometimes in general paralytics.

These examples are probably anomalies of development of the basal ganglia rather than true heterotopias. They are not in any way connected with idiocy.

Heterotopia of the cortex, however, is in more direct relation with idiocy, and is often associated with grave forms of atrophic sclerosis, microgyria from destruction, and porencephaly, and it may be with microcephaly.

But besides the heterotopias, which can be seen macroscopically, there may exist others, consisting of a few cells, which can only be recognised by the aid of the microscope.

In these cases of cortical heterotopia the cells are generally small and spherical; few are pyramidal. The larger cells are undergoing disintegration, and the smaller are in a state of atrophy.

All these forms are the result of some inflammatory process acting on the brain during embryonal life, before the cortex has completely separated from the white matter, *i.e.* before the fifth month of development.

With regard to the clinical aspect of these cases, it may be said that idiocy is always present, and epilepsy in the vast majority of instances.

In the opinion of the author, the epilepsy is due to the disturbance of the cerebral cortex, a disturbance which may not consist of macroscopic alterations of the seat of the grey substance, but rather of histological anomalies of form and arrangement of the nervous elements, anomalies by which the normal relations between these elements is completely altered.

R. G. Rows.

**SPINAL CORD DEGENERATION IN A CASE OF ACROMEGALY,  
(225) WITH TUMOUR OF THE PITUITARY REGION. ALBERT  
M. BARRETT, *Amer. Journ. of the Med. Sci.*, Feb. 1906.**

THE case reported is that of a woman aged 49, who clinically presented symptoms of acromegaly with considerable mental

change. Under treatment with thyroid considerable improvement took place, and she recovered so far as to do simple work for about 18 months. She then began to fail, became stupid and drowsy, and complained of pains in the legs and arms. The knee-jerks, which were formerly equal and brisk, became lost, and she gradually passed into a comatose condition and died. At the autopsy a large tumour was found in the region of the chiasma, filling up the sella turcica. The tumour was firmly adherent to the adjacent dura mater, and on histological examination proved to be a sarcoma.

In the cervical region of the spinal cord, degeneration of the posterior column was demonstrated both by the Weigert-Pal and Marchi method.

This degeneration does not correspond to a root degeneration, but, as the author points out, is similar to that met with in cases of cachexia and anæmia. The possibility of the degeneration being associated with the presence of other tumours in the brain is discussed, but the author concludes that the form of degeneration here met with is not that which occurs in cases of cerebral tumour. The author mentions four other cases of acromegaly in which degeneration of the posterior column has been recorded.

We quite agree with the careful wording of the title of the paper, and should be inclined to regard the spinal degeneration as due to the cachectic condition of the patient and not dependent on either the acromegaly or the tumour of the pituitary region.

FREDERICK E. BATTEN.

## CLINICAL NEUROLOGY.

### THE DISTRIBUTION AND RECOVERY OF PERIPHERAL NERVES.

(226) The Erasmus Wilson Lectures. JAMES SHERREN, *Lancet*, March 17, 24, and 31, 1906.

In these lectures the distribution and recovery of peripheral nerves is studied from the clinical standpoint.

The "exclusive" supply of the nerve under consideration, that is, the area to which it alone sends fibres, is first described from instances of section verified by operation. This is compared with the "full" supply, obtained from cases in which the nerves surrounding the area supplied by the nerve whose distribution we are studying have been divided (method of residual sensibility), and also as the result of stimulation of the trunk of the nerve.

After division of a mixed nerve, such as the median at the wrist, there may be no portion of the affected territory over which the patient cannot appreciate and localise those stimuli

commonly called tactile. The stimuli usually employed, a touch with the finger, a piece of paper or a pencil, appeal to this form of sensibility to which the name of deep touch was given. The sharpness of the point of a needle, and all degrees of temperature, are unappreciated within an area which varies in each patient; this is spoken of as loss of sensibility to prick. Surrounding this territory is an area within which the patient is unable to appreciate light touches with cotton wool and temperatures between about 22° and 40° C., and fails to discriminate the points of a pair of compasses when separated to many times the distance necessary over the corresponding portion of the sound hand. Failure to appreciate these stimuli is grouped under the term loss of sensibility to light touch. The loss of sensibility resulting from division of each nerve is examined under these headings. The divergent opinions given upon the loss of sensibility resulting from division of the various nerves is commented upon and explained. An examination is made of the muscles supplied by the nerve under examination, and attention is drawn to the mistakes which so commonly arise from failure to appreciate the extent to which the movements usually performed by the paralysed muscles can be produced by others ("supplementary motility").

On these lines, the median, ulnar, musculo-spiral and its branches, the external and internal cutaneous nerves of the forearm, the brachial plexus and the roots composing it, the sciatic, external popliteal, and posterior tibial nerves are studied.

The restoration of sensibility and motility after primary and secondary suture is next spoken of. It is shown that sensory recovery follows three well-defined stages. Sensibility to prick is first restored, the area of loss of sensibility to light touch remaining as extensive and well-defined as immediately after section of the nerve. After an interval which varies with the distance from the periphery and the nerve divided, light touch gradually begins to be restored, and at the end of the second stage the part is everywhere sensitive to light touch and all degrees of temperature. The third stage consists in the perfecting of sensibility which has been restored. This is tested by the gradual restoration of the power of appreciating two points.

Muscular recovery follows a definite march, those muscles nearest the seat of the lesion first regaining voluntary power and electrical excitability. It is pointed out that in all the cases watched by the author, irritability to the interrupted current was found to be present on the date at which return of voluntary power was first noticed. This is contrary to the usually accepted teaching.

The evidence on which the doctrine of primary union rested is examined, and the conclusion drawn that this method of recovery

is impossible, and that mistakes have arisen owing to the want of appreciation of the distribution of the nerves subserving deep touch and sensibility to prick, and of failure to examine the action and electrical reactions of the muscles supplied by the injured nerve, movements such as are usually performed by the muscles supplied by that nerve being taken as evidence of its restoration. After primary suture, complete recovery—meaning perfect appreciation of all sensory stimuli, and the return of voluntary power and electrical excitability to the affected muscles—is possible, and recovery will ensue in all cases carefully treated, provided that suppuration is avoided. No instance of rapid restoration of sensibility after secondary suture occurred in the fourteen cases watched by the author, the earliest instance being thirty days after suture. After secondary suture, recovery, both motor and sensory, occupies a longer time than after primary, and complete sensory recovery is unlikely.

The bearing of the clinical phenomena of recovery upon the theories of regeneration is briefly discussed, and the opinion expressed that the evidence, so far as it is positive, is in favour of the formation of the new nerve fibres *in situ*, and that, even when it is negative, it discloses nothing against this theory. But union with the central nervous system is necessary before the development of new axis cylinders takes place. The evidence goes to show that regeneration is "peripheral," but not "autogenetic."

Incomplete division is next defined and classified. It is shown, in opposition to the usual teaching, that a temporary interruption of continuity of a mixed nerve affects sensation earlier than motion, and its effect upon sensation is often more enduring. It is only in instances of injury to nerves, division of which produces no loss of sensibility, that the effect of the injury appears to fall wholly on the motor fibres.

Electrical reactions are described, which are considered by the author to be typical of incomplete division, as met with by the surgeon. The muscles do not react to the interrupted current, but react in a striking manner to the constant. There is increased irritability, the muscles react to a smaller current than those of the sound side, the contraction, instead of being the long drawn-out, wave-like contraction seen when the reaction of degeneration is present, is brisk, though not the sharp twitch given by sound muscles.

During recovery all forms of sensibility return together from above downwards, this being in striking contrast to the manner of sensory recovery seen after complete section of a nerve.

In conclusion, the author maintains that the peculiarities of sensory loss mentioned in the lectures, and the method of recovery, are explained by the theory brought forward in conjunction with

Dr Head.<sup>1</sup> In this communication the view was expressed that the afferent fibres in a peripheral nerve could be divided into three groups—those subserving deep sensibility, the protopathic, and the epicritic.

The distribution of the various nerves is illustrated, and tables given of the recovery after secondary suture, and of cases of incomplete division.

AUTHOR'S ABSTRACT.

**ON TABES DORSALIS.** The Lumleian Lectures. DAVID FERRIER, (227) *Brit. Med. Journ.*, March 31, April 7 and 14.

THE aim of these lectures is "to present . . . the evolution of our knowledge of tabes, its nature and causes, and to indicate the problems as yet unsolved and on which we need further light."

After a historical sketch, clinical and pathological, the relation of the posterior roots to the posterior columns of the spinal cord is described in some detail, and a diagram is given showing the constitution of the posterior columns in relation to tabes. The degenerations, characteristic of tabes in its different stages, are considered, emphasis being laid on the fact that the medium fibres of the posterior roots, *i.e.* the reflex collaterals and the fibres to Clarke's column, suffer first and most. Although the tabetic degeneration would appear to be selective in character, Ferrier does not think there is satisfactory proof that it coincides precisely with any of the foetal systems described by Flechsig and others.

According to the most modern research, the essential character of the tabetic process is a progressive dystrophy or demyelination and ultimate destruction of the nerve fibres, with secondary proliferation of the neuroglia.

**PATHOGENESIS.**—Ferrier passes in review and criticises in considerable detail the different views regarding the pathogenesis of tabes: (1) The primary ganglionic origin; (2) the peripheral origin (*v.* Leyden and Goldscheider); and (3) the meningitic origin (Nageotte, Redlich and Obersteiner). His conclusion with regard to these theories is that "there is not one which is not open to several more or less serious objections." The negative result of antisyphilitic treatment is against any theory of syphilitic meningitis, general or special (*cf.* Marie and Guillain's theory of syphilitic affection of the posterior lymphatics); and the lymphocytosis of the cerebro-spinal fluid cannot be adduced as a conclusive argument in favour of the meningitis, as the lymphocytosis has been found in other affections in which meningitis plays no part, *e.g.* Landry's paralysis, herpes zoster, the subacute combined degeneration of pernicious anæmia. Ferrier himself "is inclined to adopt the

<sup>1</sup> *Brain*, Summer Number, 1905.



hypothesis of Thomas and Hauser that the essential lesion of tabes is a dystrophy, similar to that induced by certain toxic agents, affecting the sensory protoneurone as a whole, and manifesting itself in degeneration both of the peripheral and central terminations, of which the intramedullary are the more vulnerable, and are usually the earliest to exhibit anatomical change. The process, however, is not confined to the spinal protoneurone, but may affect, among others, the optic, the sympathetic, and certain motor neurones."

"The most probable pathogeny of the tabetic degeneration is that it is the result of a toxin generated or conditioned by the syphilitic virus." Arguments, based on the statistics of Erb and Hirschl and on other facts, are quoted, which leave in Ferrier's opinion "little room for doubt that tabes and general paralysis are in all cases syphilitic, and that tabes *per se* is as much a proof of syphilis as a gumma of the skin." The relative infrequency of tabes among those who have suffered from syphilis is no argument against the syphilitic origin of tabes, but it suggests that, in addition to syphilis, there may be some predisposing or other co-operating causes. Among these, of chief importance is placed fatigue or over-exertion; the influence of sexual excess and cold has been greatly over-estimated; the same is true of neuropathic inheritance, with tendency to premature decay of nerve elements; in certain instances—perhaps five per cent.—tabetic symptoms have first made themselves manifest after injuries, but there is no proof of the efficacy of trauma as a cause of tabes independently of antecedent syphilitic affection.

In Ferrier's opinion, then, tabes is most probably due to a toxin of syphilitic origin. The reason for the special vulnerability of the sensory protoneurones to this toxin is "a question for the future"; it may be found in evolutionary or developmental causes, in peculiarities of the lymph circulation in the posterior roots and posterior columns, in an elective affinity of certain poisons for special structures, in the fact that toxins originating in the periphery ascend to the spinal cord more readily by the posterior than by the anterior roots (Homen, Orr and Rows).

It is probable, in Ferrier's opinion, that the absence of the neurilemma cells is the cause of the greater proclivity to degeneration of the intra-medullary terminals than of the other branches of the sensory protoneurone.

Degenerations, very similar to those of tabes, have been shown to occur from various poisons (*cf.* ergotism, pellagra, lathyrism; certain inorganic poisons; the toxins of diphtheria, beri-beri, etc.; certain cachectic states of the system, *e.g.* diabetes, pernicious anæmia). The resemblance between these degenerations and those of tabes "strongly supports" the hypothesis that the tabetic

degeneration is due to the action of a toxin of syphilitic origin. While this is "the most probable pathogeny of tabes," it is at present "a pure hypothesis," and one which has to surmount many difficulties, notably two: (1) The long interval between the date of syphilitic infection and the development of tabetic symptoms. In this connection, Ferrier records the interesting observation, based on examination of a series of cases, that there is no lymphocytosis of the cerebro-spinal fluid in the secondary or tertiary stage of syphilis, *i.e.* before the actual commencement of the tabetic process. (2) The fact that the tabetic process, once begun, is essentially of a progressive character, and postulates a more or less continuous generation of the poison. To meet these difficulties, Ferrier suggests that "in the absence of any living organism capable of generating the tabetic toxin"—and he gives reasons for concluding that the diphtheroid-bacillary origin of the tabetic poison (Ford-Robertson) is "at least not proven"—"it may be that the syphilitic virus under certain conditions so affects some viscus or gland that in time it develops, and continues to elaborate, some toxic internal secretion which exerts its noxious influence on the nervous system."

The third lecture deals with the *physiological pathology* of ataxy and the tabetic pupil:—

ATAXY.—"If it is possible to give a satisfactory explanation of tabetic ataxy by the demonstrable lesion of the centripetal paths to the spinal, sub-cortical (mesencephalic and cerebellar) and cortical centres, it would seem unnecessary, with Erb and others, to ascribe the ataxy to lesion of some hypothetical centrifugal tracts of motor co-ordination."

Ataxic symptoms, indistinguishable from those of true tabes, may result from lesion of the sensory nerves alone (*cf.* pseudotabes from alcohol, diphtheria, etc.), and experimental section of the posterior roots causes very similar symptoms.

There is no constant relation between the ataxy of tabes and the extent to which cutaneous sensibility, epicritic or protopathic, is affected—this form of sensibility may even not be found affected at all. "Deep sensibility" (from muscles, tendons, joints), is more important: from the result of examination of fifty cases of tabes, however, Ferrier questions the accuracy of Frenkel's contention that, in every case of ataxy, there is a more or less extensive impairment of the sense of passive movements of the joints. Sense of vibration of a tuning-fork, frequently impaired or lost in tabes, is probably dependent principally on the deep sensibility. Golla has shown that the sense of tonic or tetanic muscular contraction induced by the faradic current is greatly diminished or totally lost in the ataxic limb, while the sense of minimal or rapid contraction may be well preserved. That "total interruption of

the spinal paths of conscious sensation does not of itself cause ataxy proper" is shown by a case of syringomyelia recorded by Schüppel: the phenomena of functional cerebral anaesthesia and of sleep-walking point in the same direction.

The general result of this analysis is "that tabetic ataxy depends on impairment or loss of centripetal impressions of all kinds, conscious as well as unconscious, and, of these, those from the deeper structures (muscles, tendons, joints) are more important than the superficial; and if one may single out one class as more essential than the others of the complex, I would specify these to the spinal, sub-cortical, and cerebellar centres." Kinæsthetic impressions to these centres are necessary to secure the due co-operation of the synergic, antagonistic, and collateral muscles with the prime movers in any given movement: the constitution and connections of the spinal and sub-cortical centres provide for the combined action of these various muscles in our acts, simple and co-ordinated—"the mechanism is not formed by conscious activity, it already exists." Loss of the centripetal impressions, arising chiefly in the muscles themselves and acting on the spinal and sub-cortical (cerebellar) centres through the reflex collaterals of the posterior roots to the anterior cornua and through the fibres to Clarke's columns, is the basis of the muscular hypotonia, which is so largely responsible for many features of tabetic ataxy, *e.g.*, the attitude, the brusque over-action of the prime movers of the limbs as in walking, etc. Throughout all the acts of a tabetic with ataxy, the same purpose runs—"the attempt, by conscious effort, to make up for the failure of a self-adjusting mechanism which, under normal conditions, works better without conscious interference."

**THE TABETIC PUPIL.**—"A permanent isolated reflex iridoplegia occurs only in tabes, general paralysis, and as a consequence of congenital or acquired syphilis." The pathology of the Argyll-Robertson pupil is a subject of great difficulty. A review is given of our knowledge respecting the reflex pupillary arc and of the various theories which place the lesion underlying the Argyll-Robertson pupil in the region of the corpora quadrigemina (*e.g.*, Meynert's fasciculus retroflexus), in the central grey matter of the aqueduct of Sylvius, and in the cervical region of the cord or spinal end of the medulla. Ferrier concludes from this review that "though theoretically reflex iridoplegia may be explained by interruption of certain hypothetical centripetal paths of the light reflex, no actual pathological changes have as yet been demonstrated in the reflex iridoplegia of tabes."

The investigations of Marina, showing degeneration of the ciliary ganglia and short ciliary nerves in all cases of tabes and general paralysis with Argyll-Robertson pupil, are "of the utmost importance in reference to the pathology of the tabetic pupil."

The researches of Piltz are of great significance in the same relation; by irritation of the ciliary nerves experimentally, Piltz produced irregularities in the outline of the pupil and in its position very similar to those exhibited in tabes and general paralysis. The atrophy of the iris, too, as shown by its thin inner margin, is "undoubtedly due to affection of the short ciliary nerves." In Ferrier's opinion "it is difficult to conceive how such irregularities of the pupils could be produced by any central lesion; and seeing that they so often precede the reflex iridoplegia of tabes, the presumption is strong that both phenomena have essentially the same anatomical basis, namely, degeneration of the ciliary ganglia and short ciliary nerves." The loss of dilatation to sensory or psycho-sensory stimulation may be produced in the same way. The retention of contraction on accommodation may be explained by the assumption—in which "there is nothing improbable"—that "in tabes the ciliary ganglion and ciliary nerves are so affected that, though they cannot transmit the reflex impulse of light to the sphincter pupillæ, they can readily allow the more powerful stimulus associated with accommodation to pass through." Finally, "it is probable that the myosis . . . is due to degenerative changes of an irritative character which are going on in the sphincter."

Space forbids a more exhaustive review of the luminous details of criticism which specially mark these lectures: many important points—not directly arising from the aim of the lectures—are touched upon, *e.g.*, the importance of thorough treatment of the primary syphilis and the therapeutic possibilities of the future. Regarding nomenclature, Ferrier would like—but for their consecration by custom—to discard the terms dorsalis and tabetic, in favour of tabes simply and tabid; while, in view of "the essential pathological identity of tabes and general paralysis," it "would conduce to a truer conception of the pathology of these affections" if we were to call them spinal tabes, cerebral tabes, or cerebro-spinal tabes, according as the disease affects the spinal cord, the brain, or both conjointly.

An extensive bibliography is appended.

A. W. MACKINTOSH.

#### **THE DYSTROPHY OF TABES AND THE PROBLEM OF TROPHIC**

(228) **NERVES.** A Lecture by SIR W. R. GOWERS. *Brit. Med. Journ.*, June 2, 1906.

THE features and distribution of the arthropathy of tabes were described and illustrated by examples, and the cutaneous and other nutritional disturbances were referred to. The term "tabetic

dystrophy" has been employed, and is a convenient general designation to include them all. The risk that the real nature of tabetic arthropathy may be overlooked is definite, because it is rare even in tabes, which itself is an uncommon disease, absolutely considered. Many practitioners meet with only two or three cases of tabes throughout life, and arthropathy does not occur in more than 10 per cent. Following an injury, as it often does, the arthritis alone may attract attention, but the absence or slowness of pain should always arouse suspicion. The back-bent knee (*genu recurvatum*) must be distinguished from arthropathy. It depends only on the yielding of the ligaments through the undue strain occasioned by the unequal and changing support given by the muscles, whose tendons afford a large amount of support to the joint. The occurrence of similar dystrophies in congenital defects of development of the spinal cord was illustrated by a remarkable case in which they were associated with analgesia in one leg, which had necessitated amputation of all the toes. The nature of the case was shown by a huge scar in the lumbar region, where a long-haired mole had been removed in early life.

Trophic nerves and centres have been widely assumed to explain such disorders of nutrition. But no direct evidence of them, or of their loss, has ever been discerned. Is their assumption necessary? May not the changes depend on disease of the nerves of ordinary function? Trophic nerves were once invoked to explain muscular atrophy, but this is now known to depend on disease of the motor nerves and cells. Division of a motor nerve causes very slow changes in the muscle, with long preservation of its excitability to voltaism. An artificial irritant, exciting inflammation of the nerve, causes an irritant degeneration with rapid granular degeneration of the muscle-fibre, and in a week or two even voltaic excitability becomes extinct, never to be regained. This is seen also in some cases of intense polio-myelitis. The character of the inflammation of the nerve is so invasive as to spread to the muscular protoplasm, although the nerve-ending is not in actual continuity with the muscle substance. Thus we have evidence of an inherent vitality of the muscle, and also that it may be destroyed by extension to it of an acute change in the nerve.

All other structures (besides the viscera) are under the nutritional influence of the sensory nerve fibres. The cornea teaches the same lesson as the muscle. The vitality of the fibres depends on the ganglion of the fifth. A lesion behind the ganglion seldom affects the cornea—only when intensely irritant and spreading. When the ganglion is removed for neuralgia, with strict antiseptic precautions, the degeneration of the fibres is simple, not irritant. Yet, during the first fortnight, keratitis occurs with great readiness. The cornea has lost sensibility, and particles of dust falling on it

do not excite movement of the lids to remove them. But if the lids are fixed together, the cornea is preserved from irritation, and does not become inflamed. After a fortnight it may be exposed with impunity, though still insensitive. The nutritional instability exists only during the changing state of the nerve, and afterwards the inherent vitality of the tissue maintains its integrity. So in other tissues, affections of the nerves, as inflammation, renders the nutrition of the tissues readily disturbed. In some forms of myelitis an irritant inflammation seems to pass down the posterior roots, to involve the ganglia and descend the nerves, rendering it most difficult to preserve intact the nutrition of the skin. Thus the vital state of the nerves seems to be communicated to the related tissues, and to pervert their inherent vitality and nutritional power. The strange perverted nutrition of tabes may be thus due to the altered vitality of the nerves, which is a certain fact, without assuming the existence of wholly hypothetical trophic nerves. Under the influence of the altered state of the nerves (shown by the defective sensation), a morbid tissue process, however excited, is perverted, and the perversion persists until ultimately a vast degree of disturbance is attained. The affection of the joints usually begins as slight inflammation, often of traumatic origin, and might subside did not the absence of pain entail the absence of the needful rest. The altered process goes on in morbid form, until extreme changes result from a morbid tendency which may be in itself trifling. So with the cutaneous ulcerations. They will heal if rest is insisted on in the early stage. The inherent vitality of the tissues is sufficient, if the deranging nerve state is not supplemented by harmful physical influences.

It was also urged that the imperfect growth of the bones in infantile palsies, whether spinal or cerebral, occurs in disease of such different seat as to be irreconcilable with the hypothesis of trophic nerves. It is proportioned to the early age at which the affection occurs, and may be a simple effect of the defective approximation of the epiphyses due to the palsy and other secondary local consequences of disease.

AUTHOR'S ABSTRACT.

#### **PARALYSIS OF THE EXTERNAL BRANCH OF THE SPINAL**

(229) **ACCESSORY IN TABES.** (*La paralysie de la branche externe du spinal dans le tabes.*) Mlle. ANNA AVDAKOFF. Rousset, Paris, 1906.

THIS thesis is based on the study of a case of tabes in the service of Pierre Marie at the Bicêtre. The condition in question is uncommon, or at any rate has been rarely recorded, for in the whole of medical literature the author has only been able to dis-

cover ten other cases. The clinical study of paralysis of the spinal accessory is complicated by the fact that the sterno-mastoid and trapezius muscles receive additional nerve supply from the cervical plexus. The extent of the paralysis of these two muscles is extremely various: one or both may be affected partially or entirely, and the condition may be unilateral or bilateral. The internal branch of the spinal accessory is almost always affected at the same time, with consequent acceleration of the pulse, laryngeal paralysis, and difficulties of deglutition. There is usually also an accompanying paralysis of other nerves. Paralysis of the spinal accessory alone only occurred in four of the eleven recorded cases: in the other seven symptoms of affections of other nerves were present, the most important being the optic, oculo-motor, trigeminal, abducent, glosso-pharyngeal, pneumogastric, hypoglossal, and phrenic. Although exact post-mortem evidence is wanting, it is almost certain that the paralysis is due to a chronic meningitis affecting the nerve-roots, and not to a nuclear lesion. The case recorded by the writer is that of a man aged 42 years, who acquired syphilis at the age of 25, and had been suffering from tabes for several years prior to the discovery of the spinal accessory paralysis. The right sterno-mastoid was almost completely atrophied, and showed practically no response to faradic or galvanic stimulation. The right trapezius was in much the same condition, and there was probably also slight affection of the left trapezius. A detailed description, aided by photographs, is given of the effects of the paralysis on the movements of the head and shoulder and the position of the scapula. The patient also presented marked eye symptoms and other signs pointing to involvement of several cranial nerves. Summaries of the previously recorded ten cases are given, the first being one by M'Bride published in 1885.

HENRY J. DUNBAR.

**ON FIFTY CASES OF INFANTILE PARALYSIS.** E. F. TREVELYAN,  
(230) *Brit. Journ. of Children's Dis.*, April 1906, p. 135.

THE majority of cases occurred during the second and third years of life, and, curiously, girls preponderated largely over boys. One or both legs were involved in thirty cases, one arm in seventeen, an arm and leg of the same side in two, and an arm and leg of opposite sides in one. In six cases of the upper arm type, some of the small muscles of the hand were affected. Recovery might possibly be looked upon as complete in two cases, but in the others it was, as usual, only partial. There was generally a marked contrast between the extent of the early and of the residual paralysis. Separate foci of disease could only explain the residual

paralysis in at least nine cases. Massage, active and passive movements, and (if practicable) electrical treatment, are of service. Careful attention should be given to the prevention of deformities, especially during the early period of the disease. Orthopedic treatment, in its various forms, is necessary when deformities develop.

AUTHOR'S ABSTRACT.

**A FAMILY IN WHICH SOME OF THE SIGNS OF FRIEDREICH'S  
(231) ATAXY APPEARED DISCRETELY.** ERIC GARDNER, *Brain*,  
1906, p. 112.

In this paper the hereditary diseases of the nervous system which are more or less closely related to Friedreich's ataxia are discussed. Starting with the definition of the typical form of Friedreich's disease, and with an enumeration of the symptoms by which it is characterised, the writer reviews several divergent types of nervous disorder which have been found to occur in the same families with undoubted cases of typical Friedreich's disease. They are classed in three groups, namely those in which the knee-jerk is persistent or exaggerated; those cases which exhibit symptoms nearly related to those of Marie's cerebellar ataxy; and a smaller group in which symptoms of muscular atrophy are found in conjunction with those of ordinary Friedreich.

In addition to the examples quoted from former observers, the writer has had an opportunity of studying one family which presents points of interesting variation from the more ordinary types. The cases of disease were confined to the female members of the household, the mother and two of the daughters being affected, whilst the father and sons escaped. The mother exhibited a fine and rapid tremor of the hands on voluntary movement. This tremor was of the same character as that which is seen in disseminated sclerosis, but it never assumed a serious degree of development. The eldest daughter suffered from very marked spastic changes, but was free from ataxia. Her arms and hands showed a high degree of intention tremor, and both feet were in a condition of Pes Cavus. The second, third, and fourth daughters were found to be normal, with the one exception, that none of them had any knee-jerks. The fifth daughter had no knee-jerks, the feet showed an early condition of Pes Cavus, and some paresis of the internal rectus muscles of both eyes was present.

The conclusions to which the author of the paper is led are: (1) the common type of Friedreich's ataxy passes almost imperceptibly through an unbroken series of cases into a form of spastic paraplegia, in which the consequences of the lesion of the posterior columns of the cord are overshadowed by those due to implication



of the lateral tracts ; (2) the latter cases may further exhibit a combination of involvement of the lateral columns with symptoms of cerebral defect, thus forming a connecting link with the following type ; (3) there is a complex group, provisionally designated hereditary cerebellar ataxy, which consists of examples of family disease characterised by inco-ordination of voluntary movements combined with increased tendon reflexes, ankle clonus, and occasionally with intention tremor. Some cases included in this group exhibit ocular changes also, but deformity of the feet occurs with the greatest rarity, if at all. The writer's most important case—that of the eldest daughter—conforms in most respects to the spastic group, but in addition shows an involvement of centres which initiate voluntary movements on the one hand, and on the other exhibits the Pes Cavus of ordinary Friedreich's ataxy ; whilst the youngest girl appears likely to develop into a typical case of Friedreich's ataxia, although she presents, in addition to the usual symptoms, a paresis of the internal recti.

He therefore dissents from the opinion of those who would separate Friedreich's ataxy very sharply from the other forms of inherited nervous disease, and emphasises the fact that careful examination of many groups of cases may be expected to demonstrate the existence of transition-forms, bridging the gaps that exist between the more ordinarily recognised types.

HARRY RAINY.

#### **SYRINGOMYELIA WITH SENSORY DISTURBANCES OF RADICULAR DISTRIBUTION.**

(232) **ULAR DISTRIBUTION.** (*Note sur un cas de syringomyélie avec troubles sensitifs à topographie radiculaire.*) RAYMOND & FRANÇAIS, *Rev. Neur.*, March 31, 1906, p. 283.

THE interest of the case lies in the sensory changes, which concern not merely cutaneous sensations of pain and of temperature, but also that of the joints and bones, indicating a gliomatous process in the postero-lateral part of the cord, which in this particular case extends from the sacral region right up into the medulla. Nystagmus is rare in syringomyelia (20 times in 200 cases, Schlesinger). In the case reported it was a marked symptom. The alteration in sensation was strictly limited to the area from the second to the sixth cervical roots, inclusive, on both sides ; and it occurred also in the domain of the inferior maxillary nerve on both sides. The sensation of vibration was completely lost over the whole of the right arm and shoulder, whereas the defect of cutaneous sensation was confined to the outer aspect of the limb. Not a few cases of spastic syringomyelia with a strictly radicular change in sensation have been already described.

S. A. K. WILSON.

- A CASE OF POST-TRAUMATIC HÆMORRHAGE FROM THE**  
(233) **SUPERIOR LONGITUDINAL SINUS WITHOUT FRACTURE OF THE SKULL: OPERATION AND RECOVERY.**  
E. FARQUHAR BUZZARD and J. CUNNING, *Lancet*, March 24, 1906, p. 822.

A MAN of 25 fell on the back of his head, inflicting a small scalp wound. For 13 days he complained of constant and increasing headache; palpation of the skull revealed no abnormality, but the left abdominal reflex was absent, the left knee-jerk increased, and there was an extensor plantar reflex on the left side. Intracranial abscess was suspected. By the 17th day he had become drowsy, with subnormal temperature and intense headache, worst on the right side. There was cerebral vomiting, left hemiplegia, hemianæsthesia, and hemiopia, but no optic neuritis. On trephining, a large clot was found below the dura, and bleeding coming from the superior longitudinal sinus. This was stopped, and the wound subsequently closed. Four days elapsed before improvement set in, but recovery was ultimately quite complete. The skull was a very thick one, and probably a tributary vein had been torn away at its junction with the sinus.

J. H. HARVEY PIRIE.

- HYSTERICAL FEVER. (Zur Lehre vom hysterischen Fieber.)** G. v.  
(234) Voss (St Petersburg), *Deut. Zeitschr. f. Nervenheilk.*, H. 3-4, 1906, p. 167.

- HYSTERICAL FEVER. (Ueber das sogenannte hysterische Fieber.)**  
(235) AD. V. STRÜMPELL, *ibid.*, p. 281.

IN Voss's paper two cases are described with hysterical attacks accompanied by "hysterical fever," or, more strictly, elevation of the body temperature; in both all organic disease was believed to be excluded. In one case—that of a nurse—the temperature rose to 104° F. or even 106° F. The pulse-rate is noted once as being 104. During the attacks the skin, of the upper part of the body especially, became hot and dry, and the mucous membranes were injected. Simultaneous readings in the rectum and axilla showed that the temperature curves were parallel, but that of the rectum was always some tenths (of a degree Cent.) lower. The temperature is noted as having been taken by an experienced nurse.

In the second case the temperature varied from 101° F. to 103° F. for several months, the pulse being 90-106. The quantity of

urine was large, but otherwise normal. Edema of the legs and body occurred for a short time at each menstrual period. A full list of the literature accompanies the paper.

Strümpell in a short communication restates his conviction that the existence of the so-called hysterical fever must be regarded as "not proven." Very high temperatures, he says, are suspicious, and unless the measurement has been taken per rectum by the physician himself with his eye on the patient during the whole time, deception and fraud cannot be excluded. As regards long continued slight rises of temperature, he reminds one that latent tuberculosis, chronic inflammatory conditions, etc., may be present without recognisable physical signs. He describes several cases of the first class where, on taking the precaution of measuring the temperature himself, the "fever" at once disappeared, but he confesses it was not always clear how the apparent high reading of the thermometer was brought about.

J. H. HARVEY PIRIE.

**A CASE OF CONVULSIVE TIC.** (*Sur un cas de maladie des Tics* (236) convulsifs.) R. CRUCHET, *Archiv. gén. de méd.*, May 8, 1906, p. 1180.

THE case is that of a boy of thirteen who had suffered increasingly from this condition for three years off and on. There was a neurotic family history, and in his case there had been chorea (? tic) at the age of seven, with remaining blinking movements. At ten the tic commenced suddenly with echokinesia after a fright by a too dramatic storyteller, and very soon the whole body was affected with the characteristic movements, brusque, frequent, arhythmic, involuntary, irresistible, and subserving no purpose. With short intervals of improvement they had persisted from that time. There was also echokinesia and echolalia but no coprolalia. In Tourette's original description of the disease the movements are called inco-ordinate. The author considers this an improper description, and would call them rather voluntary movements made in spite of the will.

The patient's mental state was one of infantilism, but as regards causation he is inclined to put down the tic to a primary instability of the muscles, for some cases of tic have their mental condition intact, although there is more frequently some defect which aggravates the condition.

J. H. HARVEY PIRIE.

**EXOPHTHALMIC GOITRE.** W. GILMAN THOMPSON, *N. Y. State (237) Journ. of Med.*, April 1906.

A CONCISE and most instructive review of the clinical notes in forty-three cases is given with the object of "emphasising the importance of certain symptoms so commonly ignored in the description of this disease."

The administration of the Rogers Beebe cytotoxic serum is briefly referred to: a statement of the theory and results of its use is given.

Thompson's conclusions are: "(1) In addition to possessing the four cardinal symptoms of tachycardia, tremor, enlarged thyroid gland, and exophthalmus, Graves' disease is subject to exacerbations which are of a distinctly toxæmic character, with active fever, acute cardiac dilatation with murmurs, and a variety of symptoms constituting a definite clinical syndrome; (2) in some instances the most toxæmic attacks have been completely checked by the cytotoxic serum of Dr Rogers, prepared from the diseased human gland through animal inoculation; (3) in a large proportion of cases the agency which appears to initiate the acute toxæmic seizure is some inter-current mild infection, such as tonsillitis, pharyngitis, bronchitis, influenza, or similar acute ailment."

C. H. HOLMES.

**WHAT IS HYPNOSIS?** W. v. BECHTEREW (of St Petersburg), *Journ. (238) of Abnorm. Psych.*, Vol. i., No. 1, April 1906.

THE author first gives a short statement of the current views of hypnotism and of the differences between the school of Charcot and that of Bernheim; according to the former, hypnosis is an artificially produced neurosis akin to hysteria, and has three different stages. According to the latter school these stages are due to the education of the patients of Charcot's Clinic; hypnosis has no relation to hysteria, and is merely sleep induced by suggestion, and the different stages depend upon the depth of the sleep and the susceptibility to suggestion. The author favours the school of Bernheim, while he denies that all the facts can be explained by suggestion; in one of his patients, where there was no apparent suggestion, percussion of the tibial tendon threw the patient into a deep hypnotic sleep. He therefore considers hypnosis "a special modification of normal sleep, which can be induced by physical as well as by psychical means." There is little difficulty in accepting those views if one considers individual variations as to the sleep process, and the facts of natural somnambulism.

C. MACFIE CAMPBELL.

**DISTURBANCES OF BARÆSTHESIA, OR THE SENSE OF PRES-**

(239) **SURE.** [*Les troubles de la baresthésie (sensibilité à la pression).*] MARINESCO, *Sem. Méd.*, Nov. 29, 1905.

THE importance and significance of the sense of pressure, as distinct from tactile sensation, have recently been emphasised by Strümpell, and in the present paper a welcome addition is made to our knowledge of the subject. The author tested the sense of pressure simply by his fingers, although more or less elaborate forms of apparatus for the purpose have been invented. According to Strümpell, the sense of touch and the sense of pressure are quite distinct, inasmuch as in individuals with lax integuments the latter may be firmly pressed without giving rise to any sensation at all, until it becomes painful. He is inclined to associate baræsthesia with the muscular sense, as being a "deep" sensation, due chiefly to stimulation of aponeuroses and periosteum.

Marinesco has based his researches upon the examination of 60 cases, of which 25 were tabetics. In 24 of these baræsthesia was modified, and in every one the sensation of vibration (tested by a tuning-fork) was diminished also. The actual areas, however, in which the alterations occurred, did not always correspond. He was unable to trace any constant relation between the defect of baræsthesia and the degree of ataxia.

In his cases of paraplegia and hemiplegia, very varying results were obtained, but the general conclusion seemed to be that between pallæsthesia, baræsthesia, and the muscular sense, close affinities may be detected. They may very frequently be affected simultaneously, though sometimes dissociated. Hence they cannot be considered as identical, however much akin they may be, one to the other.

S. A. K. WILSON.

**CASE CONTRIBUTION TO THE STUDY OF SENSORY ATAXIA.**

(240) (*Kasuistischer Beitrag zur Lehre von der sensorischen Ataxie.*)

GOLDSCHIEDER, *Neurol. Centralbl.*, April 16, 1906, p. 338.

A WORKMAN, aged 44, a heavy drinker, was admitted for weakness and tingling in the right leg. The liver was large, but the other organs healthy. The right leg was weak, especially in the coarse movements, which were swaying and ataxic. Muscle tonus was much diminished there. The gait was swaying, and the knee heel test was performed with difficulty. Skin sensation was normal, but passive movements were not appreciated and the localising capacity was much diminished. The right peroneal nerve was tender. The knee-jerks were diminished, especially the right.

No other abnormal signs. The course was as follows. The ataxy and weakness progressed, as did the changes in the joint sensibility. Atrophy of the extensor muscles appeared, as did diminution of the sensibility to pain in the right leg. The right knee-jerk disappeared, and the left was only just present. In the left leg slight weakness appeared, with diminution of skin sensibility, but no change in the muscle sense and no ataxy. Later, slight tingling appeared in the arms, but no objective signs. Recovery was fairly complete in three months.

Thus the ataxy was to be correlated with changes in the joint sense, and not with cutaneous sensibility. ERNEST JONES.

**ON THE DORSAL FOOT REFLEX.** (*Ueber den Fussrückenreflex.*)  
(241) KURT MENDEL, *Neurolog. Centralbl.*, April 1, 1906, p. 293.

KURT MENDEL deals with this reflex, first described by Bechterew in 1901, and refers to a previous paper in which he demonstrated that the toe extension normally obtained by striking the dorsum of the foot was replaced in affections of the pyramidal tract by flexion of the four outer toes. In tabes, polyneuritis, and poliomyelitis no response was present. Amongst later writers is Graeffner, who, in 116 cases of hemiplegia, found plantar flexion in thirty-one cases; in twenty-nine of these Babinski's sign was present. Kurt Mendel has investigated the sign in a very large number of cases, with the following results. In healthy people, and also in functional diseases of the nervous system, toe extension (dorsiflexion) was the invariable rule. In cases of poliomyelitis or neuritis no response at all was obtained, and in such cases the plantar reflex also was absent; in a unilateral lesion of this nature the normal reflex on the healthy side thus contrasted with the absence of response on the other. The following table deals with eighty-five cases of organic hemiplegia and fifty-nine of paraplegia:—

		<i>Paraplegia.</i>	<i>Hemiplegia.</i>
1.	Babinski { Plantar foot reflex	29	19
2.	Babinski { Dorsal        „        (normal)	16	18
3.	Babinski { Plantar        „	6	7
4.	Babinski { Dorsal        „	8	41

Thus, of the 144 cases, in forty-eight both Babinski's and Bechterew's signs were positive, in forty-nine both were negative; in thirty-four Babinski was positive and Bechterew negative, in thirteen the reverse obtained. In these thirteen cases, some of which are quoted, the dorsum of the foot reflex was of the utmost value in establishing a diagnosis. Frequently it was

obtained only by percussing the outer half of the foot. Similar results were obtained in such conditions as Friedreich's ataxy, juvenile general paralysis, etc. The author, in conclusion, considers that the sign is of very high value. ERNEST JONES.

**ON A PECULIAR REFLEX CONCERNED WITH PLANTAR (242) FLEXION OF THE FOOT AND TOES IN CASES OF AFFECTION OF THE CENTRAL MOTOR NEURONS.** (Ueber eine eigentümliche Reflexerscheinung bei Plantarflexion des Fusses und der Zehen in Fällen von Affektion des centralen motorischen Neurons.) W. v. BECHTEREW, *Neurolog. Centralbl.*, April 1, 1906, p. 290.

A NEW reflex phenomenon is here described. If the patient's foot be fixed, and the toes passively flexed, there occurs a dorsal flexion of the foot and toes; in marked cases the knee and hip joints also flex. The plantar flexion of the toes must be carried out strongly. The phenomenon is easily obtained when present. It has the same significance as ankle clonus, Babinski's sign, Oppenheim's sign, Bechterew and Mendel's plantar flexion of the toes on percussion of the dorsum pedis, etc. It is, however, present in some instances when these other signs are abrogated by excessive spasticity. ERNEST JONES.

**ON SCHÄFER'S ANTAGONISTIC REFLEX.** (Ueber den Schäfer'schen antagonistischen Reflex.) W. LASAREW, *Neurolog. Centralbl.*, April 1, 1906, p. 291.

THE author discusses the reflex described by Schäfer in 1899. On gripping the gastrocnemius tightly in normal people a slight flexion of the great toe and plantar flexion of the foot appears, due to mechanical shortening of the tendons by the pressure. In affections of the pyramidal tract, extension of the toes occurs, which Schäfer attributed to a reflex contraction of the antagonists due to stimulation of the sensory nerves of the gastrocnemius tendon. The phenomenon is thus of theoretic interest apart from the clinical importance claimed for it by Schäfer as a localising sign in cases of deep coma.

Lasarew has found the sign present in ten cases within a month, seven being cases of cerebral hemiplegia and three of spinal paraplegia. In the latter cases the appearance of the sign was prompter than in the former. It signified always a pyramidal lesion. By careful observation, however, Lasarew demonstrated that the essential feature in the stimulation was not pressure on the

gastrocnemius, but the stroking or pinching of the skin over the muscle. In other words, Schäfer's sign is the well-known extensor response which, as Babinski has shown, can be evoked by stimulation, not only of the sole of the foot, but also of the leg, and occasionally even of the thigh. Thus the reflexogenous zone of the plantar reflex is more extensive than usually supposed.

The reviewer may point out that the sign here referred to was independently discovered and thoroughly investigated by Gordon, who originated a full discussion on the subject at a meeting of the *Philadelphia Neurological Society*, October 25, 1904.

ERNEST JONES.

**ON MACROPSIA AND ITS RELATION TO MICROGRAPHIA, (244) AND ON A PECULIAR ALTERATION IN LIGHT PERCEPTION.** (Ueber Makropsie und deren Beziehungen zur Mikrographie, sowie über eine eigenthümliche Störung der Lichtempfindung.) FISCHER, *Monats. f. Psych. u. Neur.*, Bd. xix., H. 3, p. 290.

THE term dysmegalopsia is employed to designate the condition in which the size of an object is inaccurately appreciated, a condition which occurs in two differing classes of disease. It is a frequent phenomenon in hysteria and epilepsy, and it may occur when there is defect of convergence or accommodation. The understanding of its pathogeny in the latter cases helps to explain its significance in the former.

The apparent size of an object is estimated by the retinal impression, as well as by the distance at which we think the object is situated. Objects of the same size, placed at varying distances, give us retinal pictures of varying dimensions. We estimate distance by the innervation we make to converge, as well as by the effort it requires to accommodate. The nearer an object, the stronger the impulse required to focus it sharply on the retina; and conversely the stronger the impulse, the nearer our estimation of the object's position. Hence, if for any reason an increased accommodation and convergence effort is demanded, the condition is one of micropsia, or mutatis mutandis of macropsia.

On the other hand, in some cases micropsia is associated with the apparent withdrawal of the object. Alteration of convergence is followed by dysmegalopsia in all individuals, relaxation of it producing macropsia and increase micropsia; but the apparent distance varies with the individual. This appears to depend on whether he regards the size of the retinal image or the degree of muscular effort; hence in micropsia the object may seem to some nearer, to others farther away.



Dysmegalopsia, similarly, is a question of accommodation. It is important not to forget that our estimation of an object's size and distance is not merely a question of convergence or accommodation. A bright surface appears larger than a dark one; a vertical line seems longer than a horizontal one; of two objects of equal length the slenderer seems the longer, etc.; and again, a large object seems nearer than a small one, a dark object appears farther off than a bright one, etc. These components have different values in different individuals, which helps to explain differences in the appreciation of size and distance, but they are not, in any case, so essential as the apparatus for accommodation and convergence.

In the dysmegalopsia of hysteria and neurasthenia, however, there is apparently no defect of peripheral organs, hence we assume disturbance of some cortical centre for accommodation. A hypothesis of this kind is distinctly unsatisfactory, nevertheless, and the author supports his contention of peripheral disturbance even in these functional cases by an ingenious series of experiments on an hysterical subject.

She was a twenty-two-year-old girl, liable to hysterical seizures of varying duration and typical character, which were associated with pronounced macropsia, micrographia, and a curious alteration in light perception whereby everything seemed to be dull grey.

The fact that the micrographia varied exactly with the macropsia was utilised by the writer in the following way:—During a period of macropsia a drop of a one per cent. hamatropin solution was applied to the right eye, and when the patient was tested two hours later it was found that with the right eye closed the usual micrographia was obtained; with the left eye closed the writing was distinctly larger. This was repeatedly observed. In further experiments by instillation of eserine the converse was demonstrated; the writing became even smaller when the eserine eye was employed. It must be obvious, therefore, how fallacious is the view that some one factor alone is responsible for the appreciation of size. Many factors come into play, and no one is negligible. The limitation of the attention in hysteria means that certain stimuli are not appreciated by the sensorium, and others therefore produce an unwonted effect. This is no doubt the explanation of the secondary micrographia, viz., the concentrating of the attention on the disturbance of vision, to the exclusion of the perception of the correcting sensations coming from the muscles.

The dyschromatopsia of the patient must have been of central origin: elimination of the macropsia by atropin and prisms was effected, but these failed to produce any change on the former.

S. A. K. WILSON.

**ON TRAUMATIC IMMOBILITY OF THE PUPIL.** (Ueber traumatische Pupillenstarre.) DREYFUS, *Muench. med. Wochenschr.*, March 27, 1906.

THIS is a reply to the criticism passed by Kreuzfuchs upon Dreyfus's previous article. It consists merely in a contradiction to several of the facts and opinions stated by Kreuzfuchs, who is said to have confused loss of sympathetic dilating power with reflex rigidity, since there is no true reflex on darkening. Dreyfus also gives references to three further cases of pupil immobility associated with injury of the spine.

JOHN D. COMRIE.

**ON TRAUMATIC IMMOBILITY OF THE PUPIL.** (Ueber traumatische Pupillenstarre.) KREUZFUCHS, *Muench. med. Wochenschr.*, March 6, 1906.

THE writer refers to the previous article of Dreyfus, and complains that attention has not been paid to his work upon the dilatation reflex of the pupil when darkened. His observations were made upon rabbits with divided fifth nerve and persons whose Gasserian ganglion had been removed. He has explained why some authors place the centre for the pupil reflex in the oculo-motor nucleus, others in the spinal cord, for, he says, the path of the light reflex is by the pupillary fibres, oculo-motor nucleus, third nerve, and sphincter pupillæ, while the darkening reflex (produced on covering the eye) runs by way of the fifth nerve, spinal cord, dilatation centre, sympathetic nerve, and dilator pupillæ. Dreyfus's case he explains by a destruction of the dilator centre in the cervical cord leading to loss of tonus in the dilator pupillæ, and thus to permanent myosis.

JOHN D. COMRIE.

**ON TRAUMATIC IMMOBILITY OF THE PUPIL.** (Ueber traumatische Pupillenstarre.) DREYFUS, *Muench. med. Wochenschr.*, Feb. 20, 1906.

THIS communication is designed to indicate the connection subsisting between the spinal cord and the pupillary reflex. The writer states that the third cervical segment is the part of the central nervous system usually found diseased when the Argyll-Robertson phenomenon is present, and he gives a brief history and copious bibliography of the views expressed and researches performed on this point. He states that, according to Reichardt, as

the result of examining thirty-five cords, a characteristic degeneration affecting a narrow strip of fibres between the tracts of Goll and Burdach from the second to the sixth cervical segments is associated with this ocular sign.

The writer records a corroborative case in which a healthy man, aged sixty-eight, sustained a severe accident, as the result of which complete paralysis of the extremities gradually came on, with death in three weeks. At first the eyes were quite normal, but, six days after the accident, both pupils were found myotic and devoid of reaction to light. They were not examined as regards convergence. Post-mortem the brain was apparently healthy, but there was a degeneration of the spinal cord extending from the last up to the third cervical segment, the result of fracture of the fifth cervical vertebra.

The writer does not attribute every case of reflex immobility of the pupil to this spinal lesion, but takes into account the possibility of defects at a higher level in general paralysis, cerebral syphilis, etc.

JOHN D. COMRIE.

#### ON TRAUMATIC REFLEX IMMOBILITY OF THE PUPIL.

(248) (*Ueber traumatische reflektorische Pupillenstarre.*) AXENFELD,  
*Deut. med. Wochenschr.*, April 26, 1906.

THIS paper deals with the production by injuries of a condition of the pupil in which the light reaction is affected in contrast to narrowing on convergence.

Three illustrative cases of damage to the optic nerve or iris are recorded. In one case a man, aged twenty-five, received a blow upon the left eyeball so severe as to tear the choroid and cause hæmorrhage; sight was but partially impaired, though even nine months afterwards the light reaction was almost absent, while the reaction, both consensually and on convergence, remained as good as in the right eye; since motor apparatus and the reflex arc from the right eye remained good, it was evident that the defect lay in the pupillary fibres of the left optic nerve or their distribution in the affected eye. In the second case, a girl aged five suffered a crush to the head beneath the wheel of a waggon, resulting in a fracture with complete paralysis of the left third nerve and contusion of the right eye shown by a small preretinal hæmorrhage. The result, as regards the right eye, was complete loss of light reaction, though the consensual and convergent reflexes remained active. The third case, that of a man struck by a splinter of wood on the left eyeball, differed from these two; the reaction in the right pupil was in every way normal, while the left pupil was slightly oval, remained absolutely unaffected by light, contracted

slightly on convergence, and dilated freely under cocaine, thus showing that there were no adhesions; the visual acuity in both eyes was normal. In this case the change is attributed by the writer not to any defect in the optic nerve, but to a condition situated in the iris.

Two other cases of damage to the skull, followed by similar reflex immobility of the pupil, are detailed. In case four, a woman, aged twenty-five, sustained a fracture of the base with paralysis of the left external rectus muscle and complete loss of pupil reflex to light in the right eye, partial loss in the left, with retention of convergence contraction in both. In case five, a woman, aged fifty-five, fell upon the right side of the head, double vision resulting, as well as complete loss of light reflex (consensual and direct) in the right eye, while the left was unaffected. The writer discusses the site of the lesion in the last case, disapproves of the idea that the ciliary ganglion is the reflex centre, and inclines to the belief that this condition may be due to a selective damage by concussion of the reflex fibres in the third nerve.

JOHN D. COMRIE.

#### ON IMMOBILITY OF THE PUPIL IN HYSTERICAL ATTACKS.

(249) (*Ueber Pupillenstarre im hysterischen Anfall.*) BUMKE,  
*Muench. med. Wochenschr.*, April 17, 1906.

THE writer states that any doubt which may formerly have existed as to the occurrence of this phenomenon in hysterical seizures has now been quite dispelled by recent observations, to which he gives numerous references. Nevertheless he states that it does not occur with great frequency. The high importance of recognising its possibility is shown by the fact that the condition of the pupils is sometimes made a test between hysteria and epilepsy.

The writer describes the condition of immobility which may be met. It is not of the nature of the Argyll-Robertson phenomenon, but consists in a rigidity of the pupil under every sort of stimulus. One or other pupil, or both, may be contracted, and in these cases there is often strong convergence also; or, on the other hand, the pupil is sometimes moderately or widely dilated.

The natural explanation in the former case is that a spasmodic condition of the sphincter iridis is present along with the spasmodic convergence; but when the pupil is dilated, opinion may differ as to whether a paralysis of the sphincter or spasm of the dilator is the cause. In a case of undoubted hysteria showing this sign, the writer was enabled, by the use of weak solutions of homatropine and cocaine, to decide in favour of a spasm of the dilator pupillæ.

JOHN D. COMRIE.

**RECURRENT THIRD NERVE PARALYSIS AS A COMPLICATION  
(250) OF ENTERIC FEVER.** (*Rezidivierende Oculomotorinalähmung als Komplikation bei Typhus abdominalis.*) JOCHMANN,  
*Deut. med. Wchnschr.*, April 19, 1906, p. 617.

JOCHMANN records a case of that interesting condition designated by Möbius recurrent third nerve paralysis, and described by Charcot under the name of ophthalmoplegic migraine. The patient was a male, aged 19, whose previous history was as follows. At the age of 8, left oculomotor paralysis occurred, ushered in by migraine, and lasted for a week. The next year he had three attacks of migraine but no paralysis. At the age of 10 he had another attack of migraine accompanied by oculomotor palsy of four days' duration. In each of the two following years he had an attack of migraine without paralysis. At 13 the third attack of paralysis took place, and like the second lasted only four days. From that date till the age of 19 he had three attacks of migraine without a return of oculomotor paralysis. At 19 an attack of migraine occurred. Diarrhoea and fever set in simultaneously. On the fifth day complete paralysis of the left oculomotor nerve developed. On the tenth day the patient was admitted to hospital, where in addition to the third nerve palsy, clinical and bacteriological evidence of enteric fever was found. This time the paralysis was of much longer duration than on each of the three previous occasions, having not completely disappeared at the end of three months. The case presented almost all the features described by Möbius as characteristic of recurrent oculomotor paralysis. The attacks occurred first in childhood. They were of longer duration than those of ordinary migraine. There were no visual aura and no hereditary nor familial history. Jochmann regards the typhoid infection as the exciting cause of the paralysis, and as responsible for its unusually long duration. J. D. ROLLESTON.

**PARALYSES OF ASSOCIATED MOVEMENTS OF THE EYES,  
(251) AND THEIR SUB-DIVISION INTO VOLUNTARY AND  
AUTOMATO-REFLEX MOVEMENTS.** (*Paralysies des mouvements associés des yeux et leur dissociation dans les mouvements volontaires et automatico-réflexes.*) CANTONNET and TAGUET, *Rev. Neur.*, April 15, 1906, p. 308.

To two cases gleaned from the literature, the authors add three fresh examples of paralysis of voluntary conjugate deviation of the eyes in conjunction with conservation of reflex conjugate deviation. The lesion is probably somewhere between the cortex and the

co-ordinating—supra-nuclear-centre, which is situated, according to some, in the anterior corpora quadrigemina, according to others in the nucleus of the sixth nerve itself. S. A. K. WILSON.

# **DIAGNOSTIC VALUE OF THE POSITION OF THE HEAD IN**

(252) **CEREBELLAR DISEASE, ETC.** (Sarcome du lobe droit du cervelet et du pédoncule cérébelleux inférieur droit. Valeur diagnostique de la position de la tête. Hypertension craniense avec hypotension rachidienne.) M. L. LARUEILLE, *Rev. Neurol.*, Feb. 28, 1906, p. 204.

THE author records the case of a boy whose head was inclined to the left shoulder and face turned towards the right. Later he had headache, vertigo, vomiting, and staggering gait. Six weeks after the abnormal position of the head was first noted, there was some cerebellar asynergy, some ataxia in all four limbs, double Babinski sign, indistinct outline of optic discs. Death occurred in the eighth week, with signs during the last fortnight of pressure on the right sixth and seventh nerves. At the necropsy a small sarcoma was found in the right inferior cerebellar peduncle, penetrating the right lobe of the cerebellum.

The case is almost identical with one recorded by Batten (*Brain*, 1903). In both the attitude is similar to that produced in dogs by extirpation of a cerebellar lobe, but on the opposite side, and in this case the side was only diagnosed when the paralytic involvement of cranial nerves occurred.

J. H. HARVEY PIRIE.

## **PSYCHIATRY.**

**TYPES IN MENTAL DISEASE.** W. A. WHITE (Washington), *Journ.* (253) *Nerv. and Ment. Disease*, April 1906.

THE problem of classification of mental diseases has been considered from nearly every possible standpoint—etiological, symptomatological, pathological, psychological, and recently from the standpoint of natural history, course and termination. Of recent years, however, the whole tendency seems to have been toward the creation of distinct clinical entities; with an increasing knowledge of the complicated functions of the brain, it is natural to expect that an increasing number of disease processes may be recognised. The accurate conception of mental disease must be from a broadly biological viewpoint; types are like species, they have many transitions and intermediate forms: as examples, the transition

forms between dementia præcox and manic-depressive insanity and involution melancholia, between paranoia and dementia præcox are mentioned. Primary confusion may occur during the course of any psychosis: flight of ideas in dementia præcox; Korsakoff syndrome in paresis and in senility; catatonic rigidity and negativism in toxic and exhaustive psychosis, etc.—these are all common examples of the complication of syndromes occurring in the so-called diseased types. Dementia should be understood to include only conditions of permanent mental impairment. White believes that with dementia as a dividing line a satisfactory classification can be arrived at until paranoia is reached. He discusses then the issue which is so commonly raised as to whether or not the degree of absurdity of a delusion represents a corresponding degree of judgment defect or dementia in the individual.

Under the *non-dementing psychoses* he includes (1) infection exhaustive psychoses; (2) toxic psychoses; (3) manic depressive psychoses; (4) psychoses associated with other diseases; (5) psychoneuroses; (6) constitutional psychopathies. Under the *dementing psychoses* he includes (1) dementia præcox; (2) involution melancholia; (3) senile and presenile psychoses; (4) paranoia and paranoid states (not otherwise classified); (5) paresis; (6) psychoses associated with other diseases.

It is to be understood in such a classification that “non-dementing psychoses may produce dementia in an unstable or a greatly predisposed individual.” “Dementing psychoses tend naturally to lead to permanent mental impairment.”

The conclusions and arguments are arranged under five heads, as follows: (1) The necessity for a broad biological viewpoint in considering the problems of alienation; (2) the inconstancy and variability of types of mental disease; (3) the desirability of a pause in the universal tendency to the analysis of mental symptoms for the purpose of developing general principles under which to group results; (4) the suggestion that a great deal could be accomplished by the study of certain symptom groups apart from the special diseases which they more or less typify; (5) the illustration of what can be accomplished by this method by its application to the dementia syndrome.

C. H. HOLMES.

#### THE RELATION OF THE PSYCHOSES OF THE INVOLUTION

(254) **PERIOD TO JUVENILE DEMENTIA.** (*Das Verhältnis der Involutionspsychosen zur juvenilen Demenz.*) G. LOMER (of Neustadt), *Allg. Ztschr. f. Psych.*, Bd. 62, H. 5, 6.

ON the basis of twenty-eight cases where the mental disorder began after 36, and excluding all cases of senile dementia, Lomer

makes a comparison of the symptoms met with in psychoses of the involution period, and those characteristic of juvenile dementia. The predominance of sexual ideas, and the similarity of the symptomatology to that of the juvenile psychosis, lead him to conclude that the involution processes and juvenile deterioration processes depend upon the same cause. He suggests that the cause is a pathological alteration of the secretion of the sexual organs modified by age and other circumstances.

C. MACFIE CAMPBELL.

**THE PSYCHOLOGY OF SUDDEN RELIGIOUS CONVERSION.**

(255) MORTON PRINCE, *Journ. of Abnorm. Psych.*, Vol. i., No. 1, April 1906.

CASES of religious conversion are classified by Starbuck into the *volitional type* and the *type by surrender*—sudden cases belong to the latter class. James believes that individuals possess large fields of subconscious thought, and in this field an incubation of motives, deposited by the experiences of life, takes place: finally these motives reach maturity and burst forth into the conscious life of the individual.

Prince advances three difficulties in the acceptance of this theory: (1) if sudden conversion is to be looked upon as a normal phenomenon, it must be proven that in normal life there is a subconscious field of sufficient size for the development of the ideas noted; (2) the experimental demonstration of any particular case is lacking; (3) there are occasional transitions of results into consciousness, for which a subconscious incubation is not easy to demonstrate.

Several cases are cited where attempts have been made to analyse the subconscious state. The final deductions are—(1) that the subconscious mind furnishes emotions rather than ideas; (2) there is no fundamental difference, except in the content of consciousness, between a state of ecstasy with its system of ideas and beliefs, and an obsession of fear or anxiety with its system of ideas and beliefs; (3) in other cases of sudden conversion the new system of ideas is not an uprush from “co-consciousness,” but rather an automatic crystallisation of past experiences out of the latest consciousness. C. H. HOLMES.

**A COMMON FORM OF INSANITY.** C. A. DREW (Mass.), *Med. (256) Rec.*, April 28, 1906.

DURING the year 1903-4 there were admitted to the hospitals for the insane in Massachusetts 2426 patients; of these, 18·7 per cent.



were classified as *dementia præcox*; 13·1 per cent. as senile insanity; 11 per cent. as alcoholic insanity; 9·6 per cent. as paresis; 5·4 per cent. as imbecility; 3·3 per cent. as epileptic insanity; 3·1 per cent. as organic brain disease.

Drew believes that *dementia præcox* is responsible for more chronic mental cripples than any three forms of insanity combined. We should be keenly alert then to recognise the danger signals; we should know its natural course and the prognosis. He criticises most pertinently the prevailing tendency to include under *dementia præcox* certain cases of manic-depressive insanity, and toxic and exhaustive cases. The percentage which recover is nearly twice as great among the relapsed cases as among those never before admitted to the hospital; this should turn our attention to the possibility of manic-depressive insanity: the prevalence of the delirium—confusion—stupor complex should at least suggest the toxic type of insanity; the overlapping of two diseased types is possible. In 454 cases of *dementia præcox* admitted to the Massachusetts hospitals in 1903-4, 16 per cent. were capable of self-support at the end of a year's time. Home environment offers great obstacles; the adolescent, who is idle, apathetic, irritable, and resistive at home, commonly falls into line at once among strangers in the routine of a well regulated hospital.

Drew's experience coincides with that of Clouston, *i.e.* that between the ages of fourteen and fifteen the liability to insanity is practically nil, that between twenty-one and twenty-five it is very great; the prognosis of psychosis developing before twenty-five is fairly good.

C. H. HOLMES.

**A STUDY OF MENTAL DISEASES ASSOCIATED WITH CEREBRAL ARTERIO-SCLEROSIS.** ALBERT M. BARRETT, *Amer.*

*Journ. of Insan.*, Vol. lxii., No. 1, 1905, p. 37.

THE author states that general paralysis, arterio-sclerotic dementia, and senile dementia are associated with characteristic changes in the central nervous system. He cites Alzheimer's four groups of cerebral arterio-sclerosis, pure types of which he says are not common. The characteristic feature of the disease in the cerebral cortex is its focal occurrence, as compared with the diffuse character of the changes in senile brain atrophy and general paralysis, and the absence of lymphoid-cell infiltration of the vessel-wall seen in the last-named affection. The patch-like appearance of nerve-tissue degeneration and its relation to the vessel changes distinguish it from the more diffuse processes of senile brain atrophy. In both general paralysis and senile brain atrophy,

arterio-sclerosis with its secondary degeneration may be present, yet it is purely associative.

The author minutely analyses a series of five cases, and summarises, to put it shortly, as follows:—In three cases there were superficial evidences of arterio-sclerosis; in four a systolic cardiac murmur with accentuated second aortic sound; and in one the heart was enlarged towards the left. Three cases of examined urine showed nephritis. They were characterised clinically by progressive dementia—loss of memory being a prominent feature—and death in coma with or without convulsions.

He very fully describes the familiar vascular changes and consequent nerve degenerations, and notes that the focal lesions were never more than 1 cm. in diameter.

He mentions many differential points clinically and etiological, such as “the lack of prominence of delusion, and a greater degree of insight into his disease in the arterio-sclerotic case; the history of vascular or similar mental disturbance in relatives, and a high hereditary taint in general paralysis, yet of a different nature.”

He admits, however, that the diagnosis clinically from general paralysis is quite impossible in a case he describes, where there was a marked arterio-sclerotic brain process, and at the same time a tabetic-like posterior column disease.

The paper is illustrated by pictures showing characteristic wedge-shaped sclerosis in cortex and degeneration in spinal cord in the case with tabes-like symptoms.

DOUGLAS M'RAE.

**ON THE PATHOGENESIS OF SOME IMPULSES.** Dr P. JANET (258) (College of France), *Journ. of Abnorm. Psych.*, Vol. i., No. 1, April 1906.

JANET gives a brief résumé of the cases of five patients with marked impulsions; these differed widely in their nature, the episodic craving being respectively for drink, food, excessive exhausting exercise, and self-inflicted pain. The features common to all the cases were the periodic appearance of the desire, the satisfaction following its accomplishment, the remorse of the subject and his good but useless resolutions. More detailed analysis of the cases demonstrated a prodromal stage, during which the patient for several days was dominated by a feeling of ennui or depression or insufficiency; it is these latter feelings which form “the essential part of the psycholeptic crisis to which the impulsions are joined only as accidental phenomena.” The author cites a case in which, owing to unfortunate associations, obsessing and impulsive ideas

of drinking became grafted on a recurrent depression. With regard to the mechanism of these impulsions, they must be regarded as an effort of the patient to escape from the primary disorder of feeling. The error of the patient is in assuming that the same means will always retain its efficiency, and that there is no other means of attaining the same end. Therapeutics should be directed to the cultivation of these other means and to the development of the internal resources of the patient. The gravity of the prognosis lies in the fact that the primary anomaly of mood is frequently constitutional and its conditions difficult to determine.

C. MACFIE CAMPBELL.

**WAS SIND ZWANGSVORGÄNGE?** Dr BUMKE, *Sammlung zwang-*  
(259) *loser Abhandlung aus dem Gebiete der Nerven- und Geisteskrankheiten*,  
Bd. vi., H. 8. Carl Marhold, Halle a. S., 1906, pp. 45.

THIS short paper is a critical examination of the many and varied phenomena described as imperative processes, obsessions, impulsive actions, and so on. The author gives a succinct account of the origins of their classification, beginning with the observations of Esquirol in 1838, and discusses at some length the respective claims to priority of their recognition and categorisation of Krafft-Ebing and Westphal—a point of purely academic interest. The author shows rightly, however, that great confusion has been introduced into this question by the opposing ways in which the term *Zwangsvorstellung* has been applied by different writers. Dr Bumke himself endorses the well-known views of Westphal, who, it will be remembered, cited four chief characteristic features, viz.: (1) intact intelligence; (2) absence of any emotional disturbance; (3) the insuppressive nature of the imperative ideas; and (4) their recognition by the subject as foreign and absurd. Dr Bumke, however, defines these phenomena in terms which he considers less liable to misinterpretation, unfortunately without any perceptible improvement. These presentations, he says, are never projected outwardly, a point of considerable differential value, but he nevertheless affirms that they do occasionally dominate the clinical picture at the commencement of the severer psychoses. He therefore dismisses as too sweeping the often-heard statement that the subjects of obsessive ideas never become insane.

Naturally, as following Westphal, he denies the emotive basis of these states. Dr Bumke traverses, with perhaps unnecessary severity, the views of Freud and his followers. The paper evidences no original work or new point of view, and is a mere criticism of the hypotheses of well-known authorities.

R. CUNYNGHAM BROWN.

- ON THE CLINICAL PICTURE OF "CIRCUMSCRIBED AUTO-  
(260) PSYCHOSIS ON THE BASIS OF A MORBIDLY DOMI-  
NANT IDEA."** (Ueber das Krankheitsbild der "zirkum-  
skripten Autopsychose auf Grund einer überwertigen Idee.")  
B. PFEIFFER (of Halle), *Monatsschr. f. Psych. u. Neur.*,  
Jan. 1906.

THE author reports the cases of two patients where, on the basis of an emotional occurrence, there developed a paranoic condition which centred round this episode without influencing the judgment of patient in other spheres. The delusions of reference and the retrospective misinterpretation of the past were limited to the same complex of ideas. These cases are similar to Friedmann's cases of mild systematising paranoia (*vide* Abstract in *Rev. Neur. and Psych.*, Vol. iii., p. 563). The author analyses the psychological mechanism of the disorder, using Wernicke's sejunction hypotheses and general terminology. C. MACFIE CAMPBELL.

- CLINICAL CONTRIBUTION TO THE PSYCHOSES OF CHILDREN.**  
(261) (*Beitrag zur Klinik der Kinderpsychosen.*) H. GOTTGRETU  
(of Münster), *Allg. Ztschr. f. Psych.*, Bd. 62, H. 5, 6.

THE author reports the case of a boy of 10 years of age who, two years after concussion of the brain with transitory delirium, began to show symptoms of mental disorder. He was restless at times; at other times he would stand staring at one spot without uttering a word. His actions were rather foolish and unexplained. He was admitted to an institution in July 1903, and there he was noted as having hallucinations both of sight and hearing. He was rather mischievous at times, sometimes was disinclined to answer questions, and after a few weeks he talked of being in America, and thought that the people were speaking American. On succeeding days he named other places. For several evenings he either saw or heard his father, and on one occasion was rather astonished over his father's presence as the journey was expensive. Patient's conduct gradually improved, hallucinations disappeared, and on November he was discharged recovered. Patient had no memory for many of the hallucinations, but had satisfactory insight for a boy of his age. For the treatment of such cases, children's hospitals or, if possible, a special division in a psychiatric clinic, would be preferable to the ordinary institution.

C. MACFIE CAMPBELL.

**TREATMENT.**

**RECENT EXPERIENCES IN THE STUDY AND TREATMENT**  
 (262) **OF HYSTERIA AT THE MASSACHUSETTS GENERAL**  
**HOSPITAL, WITH REMARKS ON FREUD'S METHOD OF**  
**TREATMENT BY "PSYCHO-ANALYSIS."** J. J. PUTMAN (of  
 Harvard), *Journ. of Abnorm. Psych.*, Vol. i, No. 1, April 1906.

PUTMAN cites in a short and clear manner the main views of Freud with regard to the mechanism of the dissociation of consciousness in certain psycho-neuroses; Freud holds that an unpleasant incident, usually of a sexual nature, becomes shut off from the rest of the personality, acts in a disintegrating way on consciousness, and that treatment should consist in the recalling of the details of the incident, and letting the patient react adequately and thus assimilate the affair. The discovery of the sore spot or hidden complex is frequently facilitated by Freud's dissociation method, and under hypnosis the patient can be brought to re-live the painful scenes. Freud's method of treatment, which consists in recalling the details of painful scenes, seems essentially different from that of authors like Dubois, who try to cultivate all associations which lead away from the disorganising element to a more rational series of associations. Putman criticises Freud's theory, and says that it is a mistake to look upon the complex of the painful incident as a fixed element in the mind; ideas are not bricks. When Freud makes his patients react adequately to a long hidden sore, he is merely asking them to associate these old painful ideas with the elements of a more vigorous and rational mental state, and therefore his method is essentially "substitutive"; it is important to recognise that Freud's method is one of substitution, and that hypnosis is not a necessary element in the treatment. One can thus avoid recalling to patient's memory unnecessary details; having got the essential facts with or without the aid of suggestion, one should cultivate in the patient's mind a truer series of associations. The author gives briefly four cases which show how, in a general hospital, suitable suggestive treatment may produce substantial results.

C. MACFIE CAMPBELL.

**THE RESULTS OF THE SURGICAL TREATMENT OF EXOPH-**  
 (263) **THALMIC GOITRE.** B. FARQUHAR CURTIS (of New York),  
*Ann. of Surgery*, March 1906, p. 335.

THIS paper reports the further progress of eighteen previously published cases of exophthalmic goitre treated operatively by the author, and records three fresh cases. The only operations requiring consideration are partial thyroidectomy and extirpation of the cervical sympathetic nerves and ganglia, ligature of the thyroid

vessels being merely a palliative or preliminary operation. In seven cases he tried sympathectomy, in the hope of avoiding the mortality from acute thyroidism, but still two deaths occurred from that condition and one from the anæsthetic. The results of sympathectomy are fairly satisfactory, but the operation is more difficult, especially with local anæsthesia, leaves a more disfiguring scar, and has as high a mortality ; the author has, therefore, returned to thyroidectomy.

Among his fourteen cases of thyroidectomy the writer has had four deaths, all from acute thyroidism. At least eight of the ten patients who recovered are "practically cured." The effect of the operation on the symptoms is interesting. The exophthalmos is lessened, though it seldom disappears, and the strained feeling in the eyes is lost. The remainder of the thyroid is stationary or diminishes, unless a relapse occurs. The heart's action generally becomes slower ; in many cases, however, tachycardia persists, but without irregular or tumultuous action, the feeling causing no inconvenience. Tremor and nervousness immediately improve, and the feeling of anxiety completely disappears.

What is the cause of acute thyroidism, which is responsible for the post-operative mortality ? The theory which ascribes it to absorption of thyroid material from the wound can for various reasons hardly be accepted. Thus the attacks occur during the usual course of the disease, apart from operation ; any excitement, fright, or worry often brings on an attack ; and, lastly, acute thyroidism occurs as frequently after sympathectomy, or after operations on distant parts of the body in patients with exophthalmic goitre, as it does after thyroidectomy. The writer believes that acute thyroidism is due to a combination of the nervous strain of operation, the effects of general anæsthesia, the shock of the operation, and the absorption of toxic material from a minimal degree of sepsis, quite as much as to absorption of thyroid products. The risk would be diminished by preliminary rest and medical treatment, by accustoming the patient to the surgeon and nurses, by the use of local anæsthesia, and by dividing the operation by performing preliminary ligation of the arteries. W. J. STUART.

#### **SERIOUS HEAD INJURIES AND THE INDICATIONS FOR**

(264) **OPERATIVE TREATMENT.** SACHS, *Boston Med. and Surg. Journ.*, Feb. 15, 1906.

**HEAD INJURIES.** MORTON PRINCE, *Ibid.*, p. 182.

(265)

**INDICATIONS FOR OPERATION IN HEAD INJURIES.** W. N.

(266) BULLARD, *Ibid.*, p. 184.

THESE three papers, which were read at a meeting of the Boston Society of Psychiatry and Neurology, repay perusal. An addi-

tional interest lies in the fact that the subject is approached from two different points of view, viz. that of the neurologist and that of the surgeon.

The following, put very briefly, are some of the conclusions. In Sachs's opinion :

1. Much more frequent collaboration should take place between the surgeon and the neurologist in cases of head injury, since, in his judgment, the difficulty of deciding whether to operate or not was much greater than the actual operation. He would, apparently, only leave the decision to the surgeon alone in severe fractures of the skull, and in clear cases of middle meningeal hæmorrhage.

2. In determining the gravity of cases, disturbances of cardiac and respiratory action, of vesical and rectal control, and the condition of consciousness were the most important points. The condition of the pupil reflexes were of no special value as an operative indication.

3. If the external injury points to one site and the symptoms to another, both should be considered, the external injury being attacked first.

4. It is unprofitable to differentiate between concussion, contusion, and compression ; rather find out whether the brain is or is not tangibly injured, and whether the injury is accessible.

5. In inaccessible cases, simple trephining may be done if increasing intracranial pressure, unrelieved by lumbar puncture, is present.

Sachs also complains that cranial surgery has not kept pace with abdominal surgery, and thinks that surgeons, by improving their technique, might produce much better results than they do.

Bullard recommends operation in the following cases, unless special contra-indications exist :—

1. All depressed fractures, and all compound fractures of the vault, even if linear and without depression.

2. In cases where cerebral symptoms are present, clearly due to traumatism—

- (a) Where unconsciousness comes on after an interval of consciousness.

- (b) Where unconsciousness has lasted for more than twelve hours.

- (c) Where persistent unilateral convulsions are present without any previous history of convulsions.

Morton Prince considers that, in the majority of cases of head injury, only general symptoms are present, and that purely "neurological signs" are rare. Consequently, the neurologist can afford the surgeon very little help. He also emphasises the frequency of laceration of the brain in severe head cases. Thus, out of 138 fatal cases examined by Dwight, in only 22 was laceration absent.

In the discussion which followed, Lund said that the differences which exist between the cranial and abdominal contents were such that cranial surgery could never be brilliant. Paralysis, also, might be produced in the course of operation, worse than the lesion which it was attempted to cure. A. A. SCOT SKIRVING.

**THE SURGICAL TREATMENT OF SCIATICA.** (Ueber chirurgische (267) *Behandlung der Ischias.*) ALFRED PERS (Copenhagen), *Deut. med. Woch.*, April 12, 1906, p. 574.

THE author believes that the operative treatment of sciatica has fallen into comparative disrepute owing to the uncertainty of its results, and this want of success he attributes to the fact that the usual method adopted is forcible nerve stretching, necessarily accompanied by injury to the nerve fibres. He recommends that the nerve should be exposed by the usual incision, but should not be stretched. It should be freed from any adhesions, and all reddened connective tissue should be very carefully removed by means of gauze and forceps from the nerve itself, so that its surface assumes its proper white and glistening appearance. The nerve is in this way followed upwards and downwards until normal portions are reached. The wound is then closed. If improvement sets in rapidly, it is ascribed to the separation of the adhesions; if more gradually, it is because "changes in the circulation have produced favourable conditions which have led to the cure of the inflamed nerve." In only two cases has the author performed this operation, and both were successful, there being no recurrence in from one to two years.

No doubt many intractable cases of sciatica are due to perineuritic adhesions, but it seems rather premature when a writer discusses the value of an operative procedure from a record of two cases.

W. J. STUART.

---

## Review

### **DIE PALPABLEN GEBILDE DES NORMALEN MENSCHLICHEN KÖRPERS UND DEREN METHODISCHE PALPATION.**

Part I. Upper Extremity. TOBY COHN, Berlin. Pp. viii. + 216, with 21 illustrations in the text. Berlin: Karger. Edinburgh: Otto Schulze & Co. 1905. Price M. 5.60.

THE student of so-called "Artistic Anatomy" is more or less familiar with the lines and points of the human body that correspond with underlying structures, of the exact nature of which



he may be ignorant. And the medical student, of course, is required to give evidence of his acquaintance with so-called "Surface Anatomy," because his ability to recognise pathological changes in the tissues of the body frequently depends on the intimacy of his knowledge of the normal, as seen and felt on the surface. The importance of surface anatomy is abundantly obvious, and it is regrettable that so little time, relatively speaking, is devoted to its study. The care with which the embryo medical dissects out the smallest ramifications of some insignificant and probably variable blood-vessel might very well be found to be more profitable were it bestowed on the landmarks of the human frame as they reveal themselves to the educated finger. Nor need the student complain of lack of material, for he always has himself to practise on, and it is indeed remarkable how much information may be gained from the systematic examination of the various regions of the body.

There is nothing new in the idea, of course: many of us acknowledge their indebtedness to such a book as Holden's "Landmarks, Medical and Surgical." The volume at present under consideration, however, is a most elaborate presentation of the structural details of the body recognisable by touch; and though it is more than two hundred pages long, it refers solely to the upper extremity. Of the wisdom of this hunting of minutiae there may be question. Many of the facts are neither of importance nor of interest, and many more are absolutely familiar. The author states in his preface that it is owing to his conviction of the desirability of being *au fait* with these details, from the point of view of scientific massage, that he has undertaken the task, which has occupied four years. But a judicious weeding out process would, in our opinion, have enhanced the value of the book. Where so many facts are aggregated, some indication of relative significance or insignificance would have been of great service. It is somewhat irritating, too, to observe the tendency to denote various points by the name of some observer: *e.g.* constant reference is made to "Mohrenheim's pit" (the deltoideo-pectoral sulcus), etc. etc. The photographs which illustrate the text are very unequal: the majority are so reproduced that they fail to justify their adoption, and constitute the best argument for the value of accurate drawings. Apart from these and other minor criticisms, one readily admits the labour and painstaking zeal that characterise the production. The arm is divided into regions, anterior and posterior, and each is exhaustively studied, from skin to bone, and every conceivable structure that can be felt is enumerated, and its relation to neighbouring tissues discussed. The book is of such a nature that further analysis becomes needless: for information regarding any particular area the original must be consulted.

S. A. K. WILSON.

# Bibliography

## ANATOMY

- PFLÜGER. Ueber den elementaren Bau des Nervensystems. Hager, Bonn, 1906, M. 3.
- SIR VICTOR HORSLEY. Note on the Tænia Pontis. *Brain*, Vol. xxix., No. 113, 1906, p. 28.
- JOHN TURNER. A Study of the Minute Structure of the Olfactory Lobe and Cornu Ammonis, as revealed by the Pseudo-vital Method. *Brain*, Vol. xxix., No. 113, 1906, p. 57.
- EDWIN BRAMWELL. The Recognition of Segmental Levels in the Cervical and Lumbar Enlargements of the Spinal Cord, from the Appearance of the Transverse Section. *Rev. Neurol. and Psychiat.*, May 1906, p. 344.

## PHYSIOLOGY

- COLIN K. RUSSEL and SIR VICTOR HORSLEY. Note on Apparent Representation in the Cerebral Cortex of the Type of Sensory Representation as it exists in the Spinal Cord. *Brain*, Vol. xxix., No. 113, 1906, p. 137.
- TOWNLEY SLINGER and HORSLEY. Upon the Orientation of Points in Space by the Muscular, Arthrodial, and Tactile Senses of the Upper Limbs in Normal Individuals and in Blind Persons. *Brain*, Vol. xxix., No. 113, 1906, p. 1.
- TAWARA. Das Reizleitungssystem des Säugetierherzens. Fischer, Jena, 1906, M. 8.
- ALRUTZ. Ueber Schmerz und Schmerznerven. *Skand. Arch. f. Physiol.*, Bd. 18, H. 1 u. 2, p. 1.
- TRENDELENBURG. Zur Frage der trophischen Nervenfunktion. *Neurol. Centralbl.*, May 1, 1906, S. 386.
- VELICH. Studien über den Einfluss des Nervensystems auf den Puls. *Wien. klin. Woch.*, May 10, p. 555.

## PSYCHOLOGY

- HOLLANDS. Wundt's Doctrine of Psychical Analysis and the Psychical Elements, and some Recent Criticisms. *Am. Journ. of Psychol.*, April 1906, p. 206.
- WHERRY. The Psychical Expression of Organic Fear. *Am. Journ. of Insanity*, 1906, p. 369.
- BORGQUIST. Crying. *Am. Journ. of Psychol.*, April 1906, p. 149.
- DUPRAT. Esquisse d'une théorie scientifique de l'activité mentale (psychogénèse). *Impr. nationale*, Paris, 1906.
- MURRAY. Peripheral and Central Factors in Memory Images of Visual Form and Color. *Am. Journ. of Psychol.*, April 1906, p. 227.
- PORTER. Further Study of the English Sparrow and other Birds. *Am. Journ. of Psychol.*, April 1906, p. 248.

## PATHOLOGY

- TAKASU. Ueber die histologischen Veränderungen der Kleinhirnrinde bei verschiedenen Nerven- und Geisteskrankheiten. *Monatsch. f. Psychiat. u. Neurol.*, May 1906, p. 458.
- GEORGE LAMB and WALTER K. HUNTER. On the Action of Venoms of Different Species of Poisonous Snakes on the Nervous System. *Lancet*, March 5, 1906, p. 1231.
- WILHELM u. ZINGERLE. Beiträge zur pathologischen Anatomie der Kretinengehirne (Schluss). *Zeit. f. Heilk.*, H. 4, 1906, p. 97.
- STRAUSSLER. Die histopathologischen Veränderungen des Kleinhirns bei der progressiven Paralyse mit Berücksichtigung des klinischen Verlaufs und der Differentialdiagnose. Deuticke, Vienna, 1906.

- FRANCESCHI. Contributo allo studio della microgria e delle vie piramidali nelle paralisi cerebrali dell'infanzia. *Riv. di Patol. nerv. e ment.*, Marzo 1906, p. 97.
- STAHLBERG. Pathologisch-Anatomische Veränderungen des Gehirns bei Lepra, Leprabacillen u. Gasserschen Ganglien, und über die Anatomie und Pathologie der Nervenzellen des Gehirns im Allgemeinen. *Arch. f. Psychiat.*, Bd. 41, H. 2, p. 596.
- R. G. ROWS. Two Cases of Embryoma in the Frontal Lobe of the Brain. *Rev. Neurol. and Psychiat.*, May 1906, p. 338.
- WESTPHAL. Ueber eine bisher anscheinend nicht beschriebene Missbildung am Rückenmark. *Arch. f. Psychiat.*, Bd. 41, H. 2, p. 712.
- KERMAUNE. Ein Fall von Spina bifida mit vorderer Wirbelspalte. *Zeit. f. Hals.*, H. 4, 1906, p. 156.

## CLINICAL NEUROLOGY AND PSYCHIATRY

### GENERAL—

- RACHFORD. Neurotic Disorders of Childhood, pp. 440. E. B. Treat & Co., New York, 1905.
- FUNCK. Radium und Nervensystem. Thieme, Leipzig, 1906, M. 0.60.

### MUSCLES—

- CAMPBELL DYKES. A Severe Case of Myoclonus Multiplex. *Lancet*, May 12, 1906, p. 1819.

### PERIPHERAL NERVES—

- SALOMONSON. Toxische Polyneuritis bei einem Phthisiker. *Neurol. Centralbl.*, May 16, 1906, S. 434.
- HARTUNG. Warum sind die Lähmungen des Nervus Peroneus häufiger als die des Nervus Tibialis. *Munch. med. Woch.*, May 15, p. 964.
- RAYMOND et BRUEL. Paralysie par Elongation du Nerf Tibial antérieur. (Soc. de neurol.) *Rev. Neurol.*, avril 30, 1906, p. 376.
- FISCHLER. Über isolierte traumatische Lähmung des N. suprascapularis und isolierte Musculo-cutaneus-Lähmung. *Neurol. Centralbl.*, May 16, 1906, S. 444.
- K. HIRSCH. Ueber einen Fall von Medianusverletzung mit seltenen trophischen Störungen. *Deutsch. med. Woch.*, May 17 u. 24.
- SICARD et BAUER. Syndrome de Landry avec réaction polynucléo-lymphocytaire du liquide céphalo-rachidien. (Soc. de neurol.) *Rev. Neurol.*, avril 30, 1906, p. 384.
- SOUQUES et VINCENT. Zona de la première racine Lombar. (Soc. de neurol.) *Rev. Neurol.*, avril 30, 1906, p. 385.

### SPINAL CORD—

- Tabes.**—MARINESCO et MINEA. Absence du Spirochaetes pallida dans le système nerveux central des Paralytiques Généraux et des Tabétiques. (Soc. de neurol.) *Rev. Neurol.*, avril 30, 1906, p. 388.
- HASKOVEC. Crises Oculaires et syndrome Pseudobasedowien dans l'Ataxie locomotrice. (Soc. de neurol.) *Rev. Neurol.*, avril 30, 1906, p. 391.
- LEENHARDT et NORERO. Sur l'état des Réflexes tendineux dans un cas d'Hémiplégie compliquée de Tabes. (Soc. de neurol.) *Rev. Neurol.*, avril 30, 1906, p. 377.
- ORSCHANSKY. Tabes dorsalis und das Kniewinkelphänomen. *Neurol. Centralbl.*, May 1, 1906, S. 401.
- ANTHONY BOWLBY. A Clinical Lecture on some Surgical Complications of Tabes Dorsalis. *Brit. Med. Journ.*, May 5, 1906, p. 1021.
- RACINE. Ueber Analgesie der Achillessehne bei Tabes. *Munch. med. Woch.*, May 15, p. 963.
- M. FAURE. Sur le traitement du tabes. *Gaz. des Hôp.*, May 15, p. 664.
- Friedreich's Ataxia.**—ERIC GARDNER. A Family in which some of the Signs of Friedreich's Ataxia appeared Discretely. *Brain*, Vol. xxix., No. 118, 1906, p. 112.
- Amyotrophic Lateral Sclerosis.**—E. MEYER. Amyotrophische Lateralsklerose combinirt mit multiplen Hirncysticerken. *Arch. f. Psychiat.*, Bd. 41, H. 2, p. 640.
- RAYMOND et GEORGES QUILLAIN. L'Amyotrophie à type lombo-pelvi-fémoral. *Press med.*, May 19, p. 317.

- Pott's Disease.**—ALQUIER. Le mal de Pott sans signes rachidiens et avec troubles nerveux. *Gaz. des Hôp.*, May 19, p. 687.
- Syringomyelia.**—RAYMOND et FRANÇAIS. Syringomyélie spasmodique avec attitude particulière des membres supérieurs. *Rev. Neurol.*, avril 30, 1906, p. 350.
- SCHLAPP. A Case of Syringomyelia with Partial Macrosmia. *Med. Rec.*, May 5, p. 702.
- Disseminated Sclerosis.**—RAECKE. Psychische Störungen bei der multiplen Sklerose. *Arch. f. Psychiat.*, Bd. 41, H. 2, p. 482.
- Hydatid.**—CONNELL. Case of Spinal Hydatid. *Inter-Colonial Med. Journ.*, March 20, 1906, p. 146.
- Cauda Equina.**—RAYMOND et ROSE. Compression des racines de la Queue de Cheval par balle de revolver. Laminectomie. Guérison. (Soc. de neurol.) *Rev. Neurol.*, avril 30, 1906, p. 381.
- Cerebrospinal Fluid.**—MERZBACHER. Die Beziehung der Syphilis zur Lymphocytose der Cerebrospinalflüssigkeit und zur Lehre von der "meningitischen Reizung." (Schluss.) *Centralbl. f. Nervenheilk. u. Psychiat.*, May 1, 1906, S. 352.

# **BRAIN—**

- Meningitis.**—BERTRAM HILL. Case of Tuberculosis Pulmonalis, with some Symptoms of Meningitis. *Lancet*, May 12, 1906, p. 1318.
- ALTMANN u. ANDERE, Arbeiten über die übertragbare Genickstarre in Preussen im Jahre 1905. Fischer, Jena, 1906.
- KÜSTER. Beitrag zur Frage des sporadischer Auftretens von Meningitis Cerebrospinalis (Weichselbaum). *Münch. med. Woch.*, 15th May, p. 956.
- SUCHY. Ein Fall von Meningitis luetica. *Wien. med. Woch.*, May 19, 1906.
- TEITELBAUM. Le Prognostic de la méningite cérébro-spinale épidémique. Firmin, Montane et Sicardi, Montpellier, 1906.
- Hæmorrhage.**—ALLAN STARR. Cerebellar Apoplexy. *Med. Rec.*, May 12, p. 743.
- Hemiplegia.**—RHEIN. Four Cases of Double Hemiplegia (Syphilitic Encephalitis) with Autopsy. *Am. Journ. of the Med. Sci.*, May 1906, p. 816.
- BOUCHAUD. Hémiplegie cérébrale infantile; réflexes abolis aux membres inférieurs, peu prononcés aux membres supérieurs. *Arch. gén. de méd.*, mai 15, 1906, p. 1236.
- Encephalitis.**—H. BEHR. Ein Beitrag zur Frage der Encephalitis subcorticalis chronica. *Monatsch. f. Psychiat. u. Neurol.*, May 1906, p. 498.
- PATOIR. Encéphalopathie atrophique de l'enfance à symptômes anormaux. *Echo méd. du Nord*, mars 4, 1906, p. 85.
- Tumour.**—P. COOMBS KNAPP. The Mental Symptoms of Cerebral Tumour. *Brain*, Vol. xxix., No. 113, 1906, p. 35.
- PARIANI. Un caso di glioma cerebrale con morte improvvisa. *Riv. di Patol. nerv. e ment.*, Marzo, 1906, p. 121.
- SCHOLZ. Einige Bemerkungen über das meningeale Cholesteatom. *Virchow's Archiv.*, Bd. 184, H. 2, p. 255.
- MINGAZZINI. Klinischer Beitrag zur Kenntnis der Hirntumoren. *Monatsch. f. Psychiat. u. Neurol.*, May 1906, p. 442.
- PÜSCHMANN. Fall von Kleinhirnbrücken Geschwulst. *Deutsch. med. Woch.*, May 24, p. 836.
- General Paralysis.**—NEUMANN. Die progressive Paralyse. Koenig, Leipzig, 1706, M. 1.20.
- VORBERG. Dementia paralytica und Syphilis. Deuticke, Vienna, 1906, M. 1.
- A. MARIE. Le Légende de l'immunité des Arabes Syphilitiques relativement à la paralysie générale. *Rev. de méd.*, May 10, 1906, p. 369.
- LÉROY. De la paralysie générale conjugale et de ses rapports avec la syphilis. Thèse, Paris, 1906.
- BABONNEIX. Les idées de grandeur dans la paralysie générale du jeune âge. *Rev. mens. des mal. de l'Enf.*, mars 1906.
- GEORGE GREENE. The Prognosis in Dementia Paralytica. *Journ. of Ment. Sci.*, April 1906, p. 284.
- Little's Disease.**—HOFFA. Die spastischen Lähmungen der Kinder und ihre Behandlung. *Deutsch. med. Woch.*, May 10 u. 17, 1906.
- Pseudobulbar Paralysis.**—RAYMOND et LEJONNE. Paralysie Pseudobulbaire chez un Enfant. (Soc. de neurol.) *Rev. Neurol.*, avril 30, 1906, p. 379.

## MENTAL DISEASES—

- BUNNEMANN. Ueber den Begriff des Psychischen. *Centralbl. f. Nervenheilk. u. Psychiat.*, May 1, 1906, S. 337.
- CLARENCE B. FARRER. The Making of Psychiatric Records. *Amer. Journ. Insan.*, Vol. lxii., No. 3, 1906, p. 479.
- G. DREYFUS. Die Inanition im Verlaufe von Geisteskrankheiten und deren Ursachen. *Arch. f. Psychiat.*, Bd. 41, H. 2, p. 482.
- OPPENHEIMER. Die medizinische Psychologie mit Bezug auf Behandlung und Erziehung der angeboren Schwachsinnigen. *Münch. med. Woch.*, May 22, p. 1023.
- ERBEN. Prüfung nervösen Störungen auf Simulation und Übertreibung. *Wien. med. Woch.*, Nos. 17, 18, 19, April and May 1906.
- WERNER. "Geistig Minderwertige" oder "Geisteskranke"? Fischer, Berlin, 1906, M. 3.50.
- STROHMAYER. Zur klinik Diagnose und Prognose der Amentia. *Monatsch. f. Psychiat. u. Neurol.*, May 1906, p. 417.
- J. SHAW BOLTON. Amentia and Dementia: A Clinico-Pathological Study. *Journ. of Ment. Sci.*, April 1906, p. 221.
- AUSTREGESILLO. Mimetismo nos Imbecis e Idiotas. *Officina de Typ. do Hosp. Nac. de Alien.*, Rio de Janeiro, 1906.
- W. A. WHITE. Etiology of Dementia Præcox. *Journ. of Am. Med. Ass.*, May 19, p. 1519.
- ARNEMANN. Über Jugendirresein (Dementia Præcox). Koenig, Leipzig, 1906, M. 1.
- W. K. WALKER. A few General Remarks regarding the Essential Nature of Dementia Præcox. *N. Y. Med. Journ.*, May 19, p. 1015.
- SANGER BROWN. Psychoses resulting from Coal-Gas Asphyxiation. *Journ. of Am. Med. Ass.*, April 28, p. 1265.
- WHERRY. Melancholia—The Psychical Expression of Organic Fear. *Amer. Journ. Insan.*, Vol. lxii., No. 3, 1906, p. 369.
- MAGALHAES LEMOS. Note sur un cas de perte de la Vision Mentale des objets (formes et couleurs) dans la Mélancolie anxieuse. (*Soc. de neurol.*). *Rev. Neurol.*, avril 30, 1906, p. 389.
- WOLFSKEHL. Auffassungs- und Makstörungen bei manischen Kranken. *Psychol. Arbeit.*, Bd. 5, H. 1, p. 105.
- SERGIO SERGI. Reflex and Automatic Excitability. *Journ. of Ment. Path.*, Vol. vii., No. 4, p. 161.
- SOUKANOFF et PETROFF. Contribution à l'étude de troubles physiques particuliers dans l'état de stupeur. *Journ. de Neurol.*, avril 25, 1906, p. 141.
- VAN HAMEL. Réforme pénale au point de vue anthropologique et psychiatrique. *Journ. de Neurol.*, avril 25, 1906, p. 148.
- ALTER. Zur Hydrotherapie bei Geisteskranken. *Centralbl. f. Nervenheilk. u. Psychiat.*, May 15, 1906, S. 394.
- MÖELI. Die in Preussen gültigen Bestimmungen über die Entlassung aus den Anstalten für Geisteskranke. Marhold, Halle, 1906, M. 1.20.
- OPPENHEIM. Psychotherapeutische Briefe. Karger, Berlin, 1906, pp. 29.
- LEROY BROUN. A Preliminary Report of Gynecological Surgery in the Manhattan State Hospital, West. *Amer. Journ. Insan.*, Vol. lxii., No. 3, 1906, p. 407.
- MAX E. WITTE. Surgery for the Relief of Insane Conditions. *Amer. Journ. Insan.*, Vol. lxii., No. 3, 1906, p. 449.
- KNAPP. Observations on some recent Surgical Cases in the Manhattan State Hospital, East. *Amer. Journ. Insan.*, Vol. lxii., No. 3, 1906, p. 467.

## ALCOHOL—

- W. FORD ROBERTSON. The Pathology of Chronic Alcoholism. *Med. Temp. Rev.*, April 1906, p. 104.
- HOPPE. Alkohol und Kriminalität in allen ihren Beziehungen. Bergmann, Wiesbaden, 1906, M. 4.
- BEVAN LEWIS. Alcoholism, Crime, and Insanity. *Journ. Ment. Sci.*, April 1906, p. 203.
- THEO. B. HYSLOP. The Vitality of a Nation. *Brit. Journ. Inebriety*, Vol. iii., No. 4, 1906, p. 156.
- A. T. SHEARMAN. The Effect of Alcohol on Feeling. *Brit. Journ. Inebriety*, Vol. iii., No. 4, 1906, p. 181.
- CHOTZEN. Ueber atypische Alkoholpsychosen. *Arch. f. Psychiat.*, Bd. 41, H. 2, p. 383.

ROEMHOLD. Ueber den Korsakowschen Symptomencomplex bei Hirnlues. *Arch. f. Psychiat.*, Bd. 41, H. 2, p. 703.

T. N. KELYNACK. The Alcohol Problem in its Biological Aspect. James, London, 1906, 2s.

FRANCIS HARE. The Medical Treatment of Inebriety. *Brit. Journ. Inebriety*, Vol. iii., No. 4, 1906, p. 196.

#### SPECIAL SENSES AND CRANIAL NERVES—

HEDDAEUS. Zur Prüfung der Pupillenreaktionen. *Centralbl. f. Nervenheilk. u. Psychiat.*, May 15, 1906, S. 385.

ALBRAND. Über wechselnde Pupillenweiten und wechselnde Pupillenungleichheit bei Geisteskranken. *Wien. klin. Rundschau*, Nr. 7, 1906.

#### GENERAL AND FUNCTIONAL DISEASES—

**Epilepsy.**—REDLICH. Bemerkungen zur Ätiologie der Epilepsie, *Wien. med. Woch.*, May 26, p. 1074.

ASCHAFFENBURG. Über die Stimmungsschwankungen der Epileptiker. *Marhold, Halle*, 1906, M. 1.60.

FERÉ. Note sur l'Épilepsie massive. *Rev. de med.*, May 10, 1906, p. 419.

EMIL REDLICH. Ueber Halbreitenerscheinungen bei der genuinen Epilepsie. *Arch. f. Psychiat.*, Bd. 41, H. 2, p. 567.

SCHISBACH. Beitrag zur Opium-Brombehandlung der Epilepsie nach Flechsig. *Arch. f. Psychiat.*, Bd. 41, H. 2, p. 684.

W. P. SPRATLING. Delayed Value of Surgery in Epilepsy in Certain Cases. *New York Med. Journ.*, May 19, p. 1025.

CHARLES K. MILLS. The Significance of Jacksonian Epilepsy in Focal Diagnosis, with some Discussion of the Site and Nature of the Lesions and Disorders causing this Form of Spasm. *Boston Med. and Surg. Journ.*, April 26, p. 453.

**Hysteria.**—KRONTHAL. Ist Hysterie eine Nervenkrankheit? *Berlin. klin. Woch.*, May 28, p. 712.

MATHIEU et ROUX. Des hématomés hystériques. *Gaz. des Hôp.*, April 26, p. 567.

A. H. GORDON. Hysterical Swelling of the Hand. *Montreal Med. Journ.*, May 1906, p. 328.

J. LÖWENTHAL. Ueber einen Fall von hysterischem Mutismus. *Wien. med. Woch.*, April 28, 1906, p. 864.

M'BRIDE. Deafness due to Hysteria and Allied Conditions. *Edin. Med. Journ.*, May 1906, p. 391.

PAUL BLUM. Des Anesthésies Psychiques dites Nerveuses ou Hystériques. *Octave Doin, Paris*, 1906, 5 fr.

MAINZER. Mitteilungen über die "Hysteria" der Tiere. *Neurol. Centralbl.*, May 16, 1906, S. 438.

ALFRED GORDON. On Auto-Suggestion in Hysteria. *Am. Journ. of the Med. Sci.*, May 1906, p. 830.

**Neurasthenia.**—W. W. KING. Tropical Neurasthenia. *Journ. of Am. Med. Ass.*, May 19, p. 1518.

TIMPANO. Neurasthenia and Neuro-Hyperasthenia. *Journ. of Ment. Path.*, Vol. vii., No. 4, p. 167.

**Tic.**—VINCENT. Les spasmes de la face (thèse). Le Bigot frères, Lille, 1906.

R. CRUCHET. Sur un cas de Maladie des Tics Convulsifs. *Arch. gén. de méd.*, mai 8, 1906, p. 1180.

NÄCKE. Nackenkrampf als Analogon zum Schreibkrampfe. *Neurol. Centralbl.*, May 1, 1906, S. 405.

BERNHARDT. Bemerkung zum Aufsatz Steyerthals über Geschichte des Torticollis Spasmodicus. *Arch. f. Psychiat.*, Bd. 41, H. 2, p. 780.

**Migraine.**—MENDEL. Die Migräne. *Deutsch. med. Woch.*, May 17, p. 785.

OTTO. Ueber einen Fall wechselseitigen Auftretens von angio-neurotischem Oedem und Migräne. *St Petersburg Med. Woch.*, No. 17, p. 181.

**Exophthalmic Goitre.**—HEINZE. Beitrag zur Behandlung des Morbus Basedowii mit Antithyreoidin Möbius. *Deutsch. med. Woch.*, May 10, p. 755.

#### MISCELLANEOUS SYMPTOMS—

J. FORTUNE. Obscure Cerebral Manifestations of Tuberculosis. *Lancet*, May 5, 1906, p. 1237.

GRASSET. Les voies de la sensibilité dans la moelle de l'homme. *Gaz. des Hôp.*, May 12, p. 651.

- KNAPP. Syphilitische Sensibilitätsstörungen am Rumpfe. *Arch. f. Psychiat.*, Bd. 41, H. 2, p. 737.
- DEJERINE. La claudication intermittente de la moelle. *Rev. Neurol.*, avril 30, 1906, p. 341.
- GRILLS. A Case of One Cerebral Hemisphere supplying both Sides of the Body. *Brit. Med. Journ.*, May 5, 1906, p. 1033.
- L. W. WEBER. Gleichseitige Krämpfe bei Erkrankung einer Kleinhirn-hemisphäre. *Monatsch. f. Psychiat. u. Neurol.*, May 1906, p. 478.
- GRASSET. Monoplégie d'origine corticale (traumatisme à la partie antéro-supérieure de la région rolandique gauche, paralysie articulomotrice de l'avant-pied et anesthésie segmentaire en bottine à droite, jacksonisme psychique et moteur). *Province méd.*, mars 10, 1906, p. 109.
- A. H. FERGUSON. Ischemic Muscular Atrophy, Contractures and Paralysis. *Ann. of Surgery*, April 1906, p. 599.
- FOERSTER. Die Kontrakturen bei den Erkrankungen der Pyramidenbahn. Karger, Berlin, 1906, M. 2.
- FRANKL-HOCHWART. Der Menieresche Symptomencomplex. Hölder, Vienna, 1906, M. 3.
- WILFRED TROTTER. The Cheyne-Stokes Phenomenon in Acute Cerebral Compression. *Lancet*, May 19, 1906, p. 1380.
- LUNDIE. Transient Blindness due to Spasm of the Retinal Artery. *Ophthalm. Rev.*, May 1906.
- PIERRE BONNIER. Inversion du phénomène de Ch. Bell chez une Labyrinthique. (Soc. de neurol.) *Rev. Neurol.*, avril 30, 1906, p. 375.
- BUMKE. Ueber abnorme Bewegungserscheinungen am Kopfe nach Schädelbasisfraktur. *Centralbl. f. Nervenheilk. u. Psychiat.*, May 15, 1906, S. 386.
- STANLEY BARNES. The Knee-jerk in Pneumonia. *Birm. Med. Rev.*, April 1906, p. 199.
- WEHRUNG. Über einseitiges Fehlen und über die Wiederkehr des verschwundenen Kniephänomens. *Neurol. Centralbl.*, May 1, 1906, S. 391.
- J. D. ROLLESTON. The Abdominal Reflex in Enteric Fever. *Brain*, vol. xxix, No. 113, 1906, p. 99.
- Aphasia.**—KRAEPELIN. Über Sprachstörungen im Traume. Engelmann, Leipzig, 1906.
- SPILLER. Lesions of the Left First Temporal Convolution in Relation to Sensory Aphasia. *Rev. Neurol. and Psychiat.*, May 1906, p. 329.
- EDWARD JACKSON. Developmental Alexia (congenital word blindness). *Am. Journ. of the Med. Sci.*, May 1906, p. 843.
- LAMY. Troubles d'élocution chez un ancien aphasique. *Soc. de Neurol.*, fév. 1906, p. 186.
- NATHAN. Note sur un cas d'amusie incomplète chez un musicien professionnel atteint également d'aphasie sensorielle très atténuée. *Soc. de Neurol.*, fév. 1906, p. 202.
- C. J. THOMAS. Cécité congénitale pour les mots imprimés. *Clin. Infantile*, fév. 1906, p. 72.
- RAYMOND et EGGER. Un cas d'Aphasie tactile. (Soc. de neurol.) *Rev. Neurol.*, avril 30, 1906, p. 371.
- PIERRE MARIE. Revision de la question de l'Aphasie: la troisième circonvolution frontale gauche ne joue aucun rôle spécial dans la fonction du langage. *Semaine méd.*, mai 23, p. 241.
- HEILBRONNER. Ueber Agrammatismus und die Störung der inneren Sprache. *Arch. f. Psychiat.*, Bd. 41, H. 2, p. 653.

#### TREATMENT—

- M. FAURE. Comment faut-il traiter les hémiplegiques. *Gaz. des Hôp.*, May 22, p. 701.
- RAUZIER. Traitement de la migraine. *Arch. gén. de méd.*, mai 1, 1906, p. 1107.
- GERLACH. Versuche mit neuronal bei Geisteskrankheiten. *Münch. med. Woch.*, May 22, p. 1017.
- BRÜGELMANN. Die Behandlung von Kranken durch Suggestion und die wahre wissenschaftliche Bedeutung derselben. Thieme, Berlin, 1906, M. 1.
- PROSCHECK. Übungssystem koordinierter Muskelgruppen. Steinacker, Leipzig, 1906, M. 5.
- ASCH. Herz- und Nervenleiden und ihre Behandlung mit unterbrochenen und Wechselströmen. Baake, Berlin, 1906, M. 0.50.
- FRAIKIN et GRENIER DE CARDENEL. Indications générales de la thérapeutique physique dans les maladies nerveuses et les maladies orthopédiques. Pariset, Paris, 1906.

# Review of Neurology and Psychiatry

---

## Original Articles

### **THE PARS INTERMEDIA OR NERVUS INTERMEDIUS OF WRISBERG, AND THE BULBO-PONTINE GUSTATORY NUCLEUS IN MAN.**

By DR J. NAGEOTTE,

Physician to the Bicêtre Hospital, Paris (Travail du laboratoire d'histologie  
de l'École des Hautes Études au Collège de France, et du service de  
M. le Dr Babinski, à l'hôpital de la Pitié).

HAVING had occasion to study, by Marchi's method, a case of degeneration of the nervus intermedius of Wrisberg in man, I have been able to determine the exact conformation and connections of the bulbo-pontine nucleus to which the fibres of this nerve belong. This nucleus measures about fourteen millimetres in length; it receives the fibres of the intermediary nerve only in its middle, most slender part; above and below it becomes enlarged in order to receive the fibres of the trigeminus and of the glosso-pharyngeal. In man, when studied in serial sections stained by the Weigert-Pal method, it presents definite anatomical characters which make it easily recognised throughout its whole extent, so that its position can be determined independently of its connections with the surrounding parts. The connections of this nucleus with the intermediary nerve of Wrisberg, which, as Sapolini has shown, is continuous with



the chorda tympani, indicate that its function is the reception of gustatory impressions; conversely, the fact that it receives a great number of fibres from the trigeminus, seems to me to prove that the fifth pair contains, in its intra-pontine course, a gustatory nerve, which is evidently the lingual.

In this article I shall describe, firstly, the degeneration of the intermediary nerve of Wrisberg in the case which I have observed; secondly, the topography and the myelinic network of the gustatory bulbo-pontine nucleus in man (making use of three series of sections, cut millimetre by millimetre, and stained by the Weigert-Pal method, also of a series stained by carmine and blue soluble in alcohol, by the method of Mathias Duval). Finally, I shall compare the results obtained with the views arrived at by anatomists who have preceded me, and shall state the observations which corroborate the anatomical and physiological deductions drawn from study of my sections. This work will have to be completed subsequently by study of the fine structure (methods of Nissl and Ramon y Cajal), and by the researches of comparative anatomy.

A man, aged 36, suffering from cancer of the stomach, presented, about a month before his death, a right facial paralysis, which was demonstrated on post-mortem examination to be caused by a metastatic nucleus as large as a grain of hemp-seed, situated in the aqueduct of Fallopius immediately *below* the geniculate ganglion. The details of the observation having been lost, I cannot say whether the patient presented any sensory troubles, but this blank, though important, may be filled up by what we know of analogous cases, in which there usually exist localised disturbances of taste.

The facial nerve, as well as the chorda tympani, on being examined in a series of transverse sections by Marchi's method, are found to be completely degenerated below the lesion; the geniculate ganglion, the extra-pontine portion of the intermediary nerve and the portion of the facial comprised between the cancerous nucleus and its emergence from the pons do not, on the contrary, present any appreciable lesions by Marchi's method. But in the interior of the pons the facial presents manifest traces of retrograde degeneration, and this degeneration becomes accentuated in proportion as one approaches the nucleus,

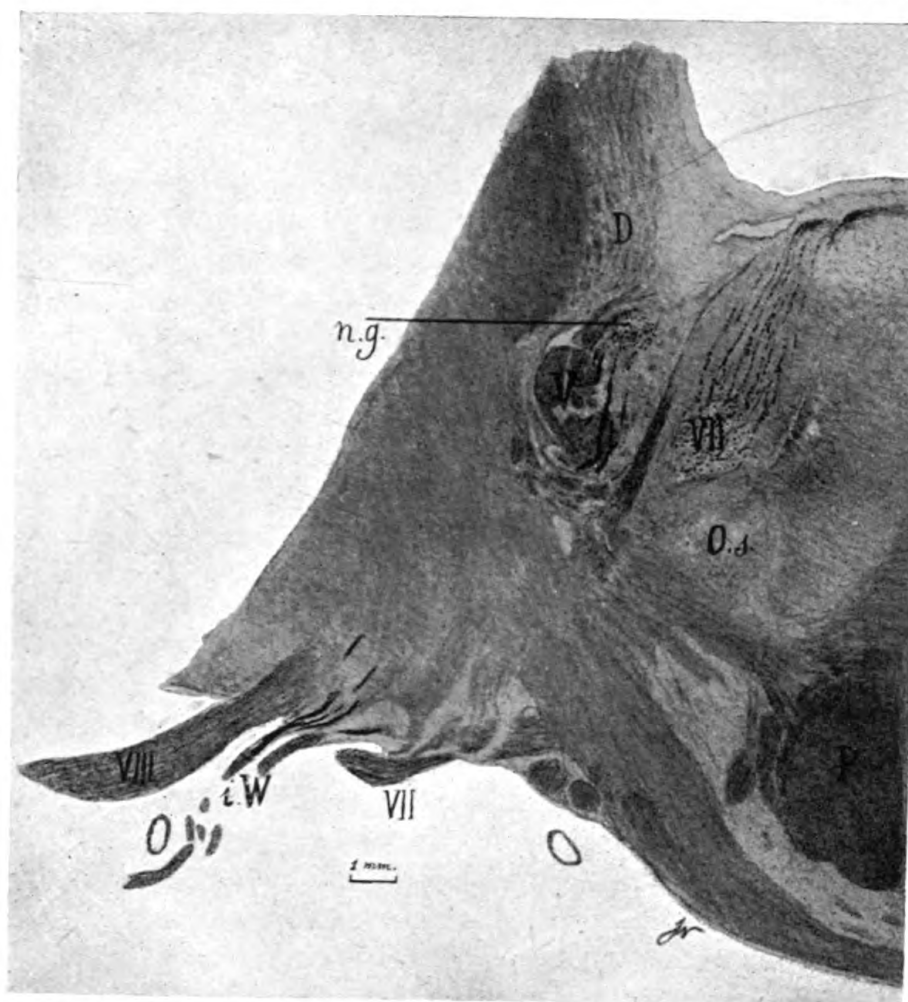
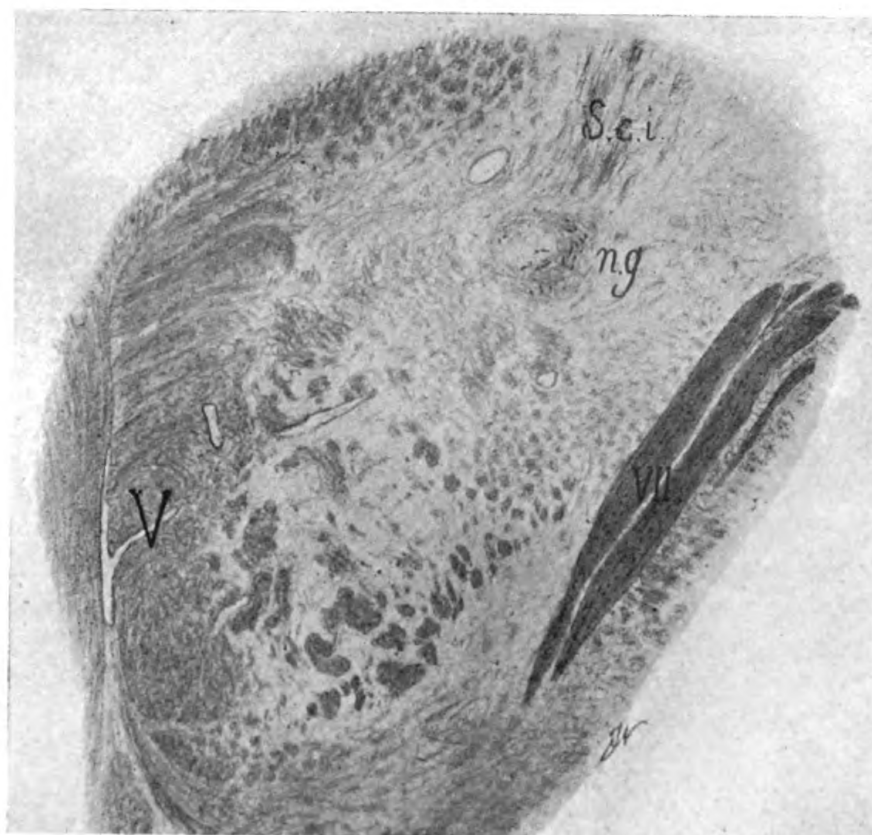


FIG. 1.





which contains a great number of degenerated fibres throughout its whole extent.

As regards the intermediary nerve of Wrisberg, its degeneration presents a very remarkable distribution. I have just indicated that its extra-pontine portion would appear to be intact; in its intra-pontine course, on the other hand, this nerve shows, by Marchi's method, a complete degeneration of its fibres. The aspect of this degeneration is identical with that of Wallerian degeneration. The line of demarcation between the intact portion and the degenerated portion is absolutely clear; it is situated exactly at the point where the root fibres normally lose their sheath of Schwann as they penetrate into the nerve centres. I should recall the fact that at this site there exists in every sensory root a space in which the myeline is paler; as all the neighbouring sheaths present this modification at the same level, the result is the formation of a bright line which cuts each root fascicle in the neighbourhood of its point of penetration. This line separates the peripheral portion, which is provided with its sheath of Schwann, from the central portion, which has lost it. This line has a form which varies in the different fascicles of the same root: sometimes it is straight, and is situated at the level of the pia-mater, sometimes it describes a very marked curve, the convexity of which is directed towards the periphery, so that each of its extremities being at the level of the pia-mater, its centre is placed one or more millimetres outside; in this case the central portion of the nerve fascicle forms a kind of tubercle protruding outwards, which is capped by the peripheral portion. In the observation in question these tubercles are degenerated, as is all the central portion of the fascicles of the intermediary nerve of Wrisberg, whilst the peripheral portion is spared. This distribution is distinctly seen in fig. 1. It is necessary to insist upon this anatomical detail in order that there may be no doubt as to the precise point at which the degeneration commences.

The particular mode of degeneration which is here described, and which is very different from Wallerian degeneration proper, is not yet known; it seems to me to present a certain interest. I cannot dwell longer upon its significance in the present article, which has quite another aim, but it is necessary to state precisely the general conditions of this phenomena as it is presented to observation in this case. A sensory root neurone, that is to say

a neurone with a single prolongation bifurcated in T-form, has recently undergone, very near the cell, an amputation of the peripheral portion of its axone. Although the cell has not been touched, the central part of the axone has undergone an intense degeneration which, by Marchi's method, appears only in the portion of its course which belongs to the central nervous system. And it should be noted that the conditions of observation are excellent; the meninges are intact, and in the whole course of the nerve in question (which has been examined after having been divided through its whole extent into a series of short lengths) there exists but the one and only cause of degeneration described above, that is to say, a nucleus of cancer situated immediately *below* the geniculate ganglion. This proves once again that a section of the peripheral axone, when made very near the cell, is capable of producing a considerable perturbation in the vitality of the neurone; in the case in question this perturbation is shown by a very marked lesion of the central axone, a lesion which is perhaps only one phase of a total destruction of the neurone. To this particular mode of degeneration one may give the name of *secondary transcellular degeneration*.

A second point is also made evident by this observation, that is the very great instability (*fragilité*) of the myeline of the central portion of the root as compared with that of the peripheral portion. In fact, whilst the former is already completely degenerated, the latter as yet shows no distinct sign of having suffered. I have had occasion to observe analogous facts under other circumstances, in particular in study of root lesions of the cord associated with cerebral tumours.

Since all the fascicles of the intermediary nerve of Wrisberg present an intense degeneration from their entrance into the pons, this case may serve, just as well as if it were one of real Wallerian degeneration, for investigation of the topography of this nerve.

*Intra-pontine course of the intermediary nerve of Wrisberg.* The fascicles which constitute this nerve, seven or eight in number, penetrate into the pons between the facial and acoustic nerves; they lie nearer to the eighth pair than to the seventh. Their course is somewhat sinuous; they cross the fibres of the pons, then those of the trapezius, in order to reach the root of the trigeminal, which they cross obliquely; finally, they terminate

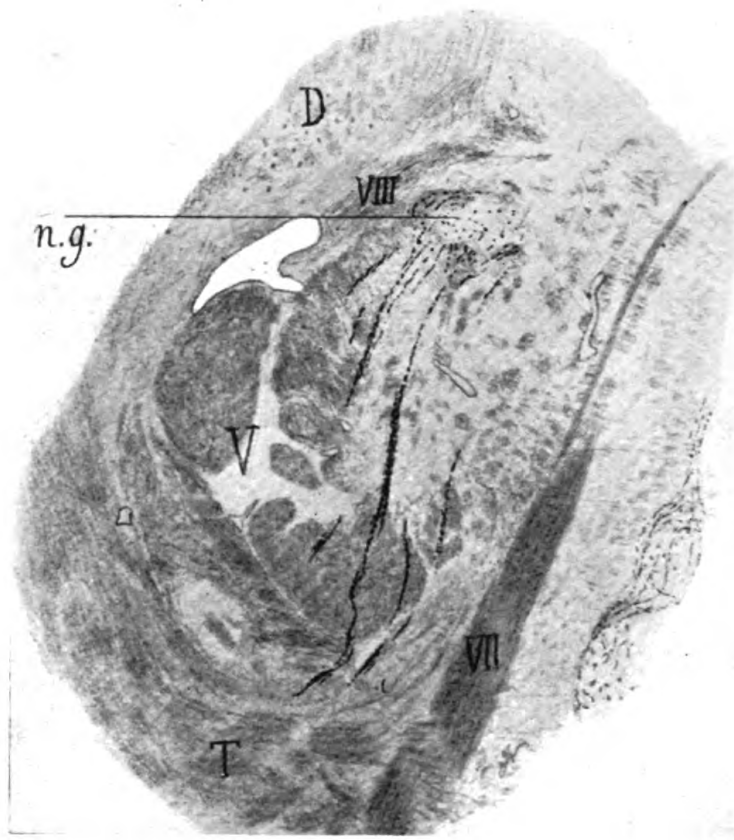


FIG. 3.

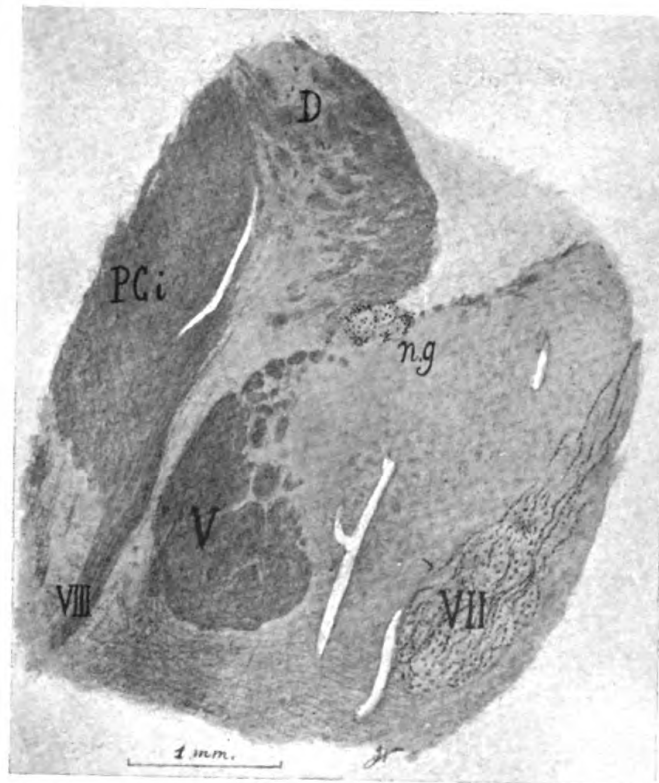


FIG. 4.



in a very limited nucleus, which is situated in the prolongation of the anterior cornu of the crescent, which is formed by the descending root of the fifth pair. As they approach the nucleus in which they terminate, they become subdivided into numerous fine fascicles, which are spread out over the root of the trigeminal, and then converge towards their point of destination. The nucleus appears in the sections of the levels under consideration, in the form of one or more small masses of grey matter, rounded in outline and enclosed in a mantle of white matter. This formation is clearly marked out and possesses a characteristic aspect; it extends upwards and downwards from the point which the fibres of the intermediary nerve reach in order to become connected above with the fibres of the trigeminal, and below with the fascicles of the glossopharyngeal. Figs. 7 and 8 represent the projection of the nucleus in question on the median plane and on an anteroposterior plane; these figures, made with an enlargement of three diameters, have been obtained by the aid of measurements taken on a series of transverse sections at a distance of 1 mm. It will be seen that the zone of entry of the fibres of the intermediary nerve measures about 2 mm. in breadth. The superior extremity of the nucleus is situated about 3 mm. above the superior limit of the zone of entry, and its inferior extremity descends about 9 mm. below the inferior limit of this zone, so that the nucleus measures in all about 14 mm. in length.

Study of sections made by Marchi's method shows in addition that the fibres of the intermediary nerve are not distributed only in the portion of the nucleus which is reached directly by the fascicles of that nerve. We find, in fact, a great number of degenerated fibres which follow an ascending or descending course in the white matter at the periphery of the nucleus, fibres which terminate in the superior and inferior regions of the grey matter. Above, these fibres, which are situated more particularly within the nucleus, diminish rapidly in number, and the last of them disappear about  $1\frac{1}{2}$  mm. from the superior limit of the zone of entrance, so that the superior extremity of the nucleus, which is enlarged, does not receive any fibres from the intermediary nerve over a space of about 1 mm. Below, the degenerated fibres diminish less rapidly; some of them descend



to  $4\frac{1}{2}$  mm. below the inferior limit of the zone of entrance, that is to say to the neighbourhood of the point at which the gustatory nucleus enters into intimate connection with the solitary bundle, as we shall see later.

To sum up, the fibres of the intermediary nerve are distributed in the gustatory nucleus over an extent of about 8 mm.; they blend above with root fibres which certainly come from the trigeminal, and below with other fibres which belong to the glosso-pharyngeal. We shall see subsequently also that certain facts tend to prove the existence of fibres from the trigeminal which descend along the periphery of this nucleus and which terminate at various points throughout its whole length. The zones of distribution of the three gustatory nerves in the interior of the common nucleus of termination thus largely overlap each other.

I ought to add that there exist root fasciculi which follow almost the same course as those of the intermediary nerve of Wrisberg, and which certainly do not belong to this nerve, or at least to its sensory part. These fasciculi are of considerable size; they are situated somewhat internally to the fibres of the nerve of Wrisberg, and like them are directed towards the gustatory nucleus; but instead of resolving into fibres which penetrate into this nucleus, they pass inside it and become lost in the grey matter of the fourth ventricle. The fascicles in the case which we are studying contain some distinctly degenerated fibres, which become separated from them in the neighbourhood of the gustatory nucleus in order to enter into this nucleus. These fibres evidently belong to the intermediary nerve. But, in addition, it would seem that even in the pure portion<sup>1</sup> of these fascicles, there exist a number of fine globules more numerous on the affected than on the healthy side. It is thus possible that we have here a retrograde degeneration analogous to that observed in the facial nerve of the same side. Reasoning by exclusion, one might suppose that these root fascicles, which I have found exactly at the same place in the three series of normal sections studied, represent the vaso-motor roots of the intermediary. In any case they occupy exactly the same position in relation to the sensory and the motor roots of the seventh pair (the intermediary

<sup>1</sup> *i.e.* Those fascicles which remain after the degenerated fibres pass off to the gustatory nucleus.



FIG. 5.



and facial proper) as do the fibres arising from the dorsal nucleus of small cells of the ninth pair in relation to its sensory and motor portions. Now, we know that Dr Alexander Bruce considers the dorsal nucleus of the ninth pair to be the origin of the vasomotor fibres of this nerve; this fact would thus constitute a fresh analogy between the ninth and the seventh pairs. It would have been interesting to determine experimentally the place of the nucleus of origin of these problematical fibres by looking for Nissl's *réaction à distance* after extraction of the facial nerve; but the only experiment which I practised upon the rabbit with this intention gave me no results.

*Gustatory Nucleus.*—We must now study more closely the topography and the structure of this nucleus.

The gustatory nucleus is situated in front of the sulcus on the floor of the fourth ventricle, which separates the fasciculus teres from the acoustic area. We know that, corresponding to this sulcus on its posterior surface, the grey matter of the floor of the fourth ventricle forms by its anterior surface a ridge which projects forwards (dorsal nucleus, triangular nucleus of the acoustic), and which penetrates somewhat deeply between Deiters' nucleus and the formatio reticularis of the pons and medulla. The gustatory nucleus, which is in the form of a column, is situated immediately in front of the crest of this ridge, as if it were a detached portion of it. Above, it commences to appear at about 2 mm below the termination of the *convolutio trigemini*; below, it ceases 3 or 4 mm. below the origin of the solitary bundle. In its inferior portion it constitutes what is now known as the nucleus or *gelatinous substance of the solitary bundle*, very exactly represented by Dr Alexander Bruce in his Topographical Atlas of the Mid and Hind Brain. In its superior half, it stands in intimate relation to the sensory nucleus of the trigeminus, from which it has not been differentiated by the classic writers, although it may be easily recognised in the good figures of this region given in their treatises. Through the whole extent of its connection with the trigeminus, the gustatory nucleus is situated exactly in the prolongation of the anterior cornu of the crescent described by the descending root. In all its length the nucleus in question is in very intimate relationship with that which M. and Mme. Déjerine call the *juxtarestiform body*, that is to say the nucleus of Deiters accom-

panied by descending fibres from the acoustic and by the internal semi-circular fibres of the cerebellum.

Above, the gustatory nucleus is in connection with the ascending (inferior) root of the trigeminus, the fascicles of which skirt its superior extremity before uniting with the descending (superior) root.

The aspect of the gustatory nucleus, in transverse sections stained by the Weigert-Pal method, is very characteristic, and permits of it being described in two portions. In its superior three-fifths, it is formed by one or more small rounded masses of grey matter, surrounded by a layer of white matter; in its inferior two-fifths, on the contrary, it is broken up into several irregular portions by white fascicles, which tend to lie in its centre, and which come from the glosso-pharyngeal (figs. 5 and 6, sections 398, 360, 350, and 337). The first portion of the nucleus is formed, to begin with, by an enlarged part, which measures on an average 0.8 mm. in diameter, and which constitutes about one-third of the total length; below, comes a very much thinner portion, in which the grey matter hardly measures 0.1 mm. in diameter, and even seems in certain individuals to be interrupted in places, the continuity of the nucleus being in this case established only by its white matter, which always retains a very characteristic aspect. Finally, the nucleus becomes swollen shortly before the termination of its first portion, which is marked by the arrival of ascending bundles from the glosso-pharyngeal. The penetration of the fibres from the intermediary nerve takes place half in the inferior extremity of the superior enlargement, half in the narrowed part which succeeds it. (Fig. 6, sections 326, 314, 302, 278, and 266.) In its second portion the characteristic appearance of the gustatory nucleus is completely transformed by the penetration of fibres from the glosso-pharyngeal. The white mantle which surrounds the grey matter disappears. The latter is divided into small areas by the sensory root-bundles of the ninth pair, some of which ascend into the body of the nucleus and go to reinforce the peripheral white matter of the inferior regions of the first portion of the gustatory nucleus (cerebral prolongation of the solitary bundle), whilst the others assume a descending direction and go to constitute the solitary bundle proper. When the solitary bundle is formed (fig. 6 section 302),



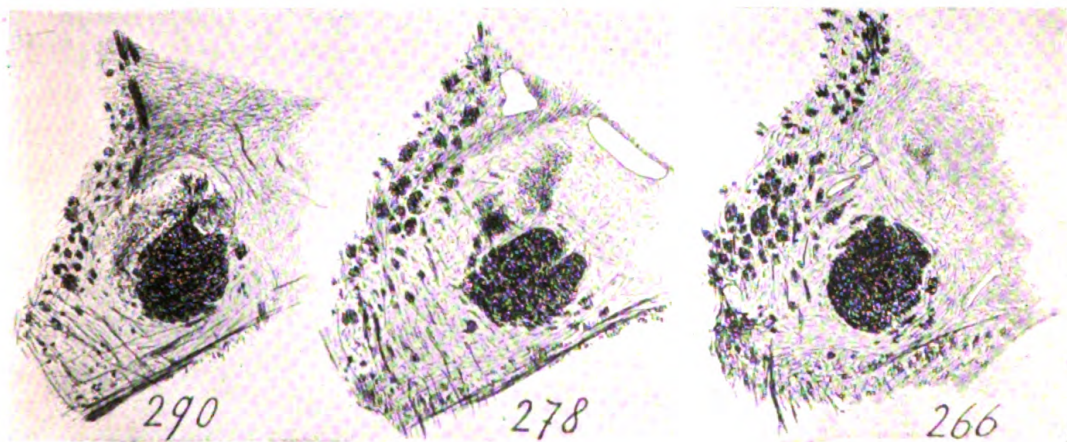
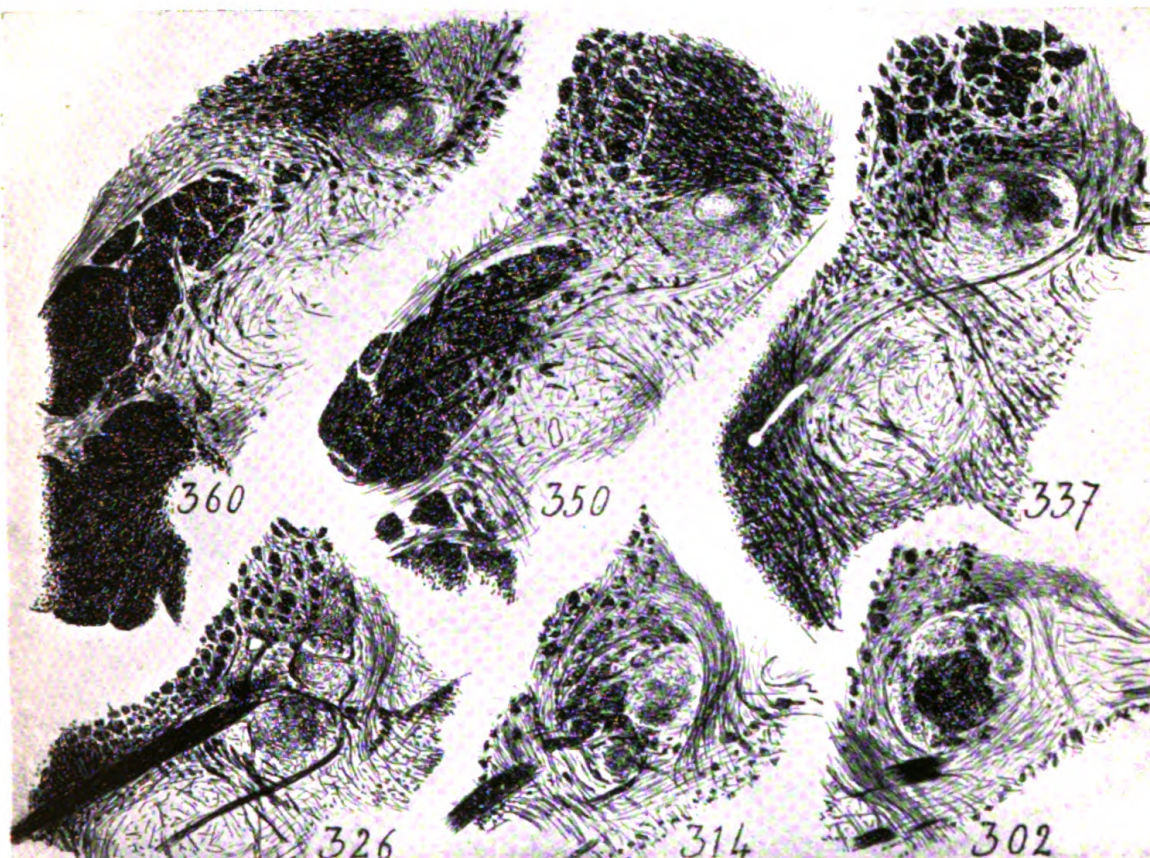


FIG. 6.



the grey matter of the gustatory nucleus surrounds it with an almost complete ring, which is thicker posteriorly; then the anterior part of this ring disappears and there remains only a posterior crescent. Finally, the nucleus, which diminishes rapidly in size, separates itself from the solitary bundle behind, in order to plunge into the grey matter of the floor, retaining its limits perfectly distinct, and terminating in an attenuated extremity at about half a millimetre from the posterior margin of the solitary bundle. The remarkable conformation of the *gelatinous substance of the solitary bundle* in man, and the termination of this nucleus in an extremity which curves backwards has never been described in detail to my knowledge. This arrangement, however, is probably constant; I have found it, without any appreciable variation, in the three series of sections at my disposal. The inferior portion of the nucleus which curves backwards, and which forms as it were an appendix to the grey matter of the solitary bundle, is perhaps the vestige in man of the *commissural ganglion of Cajal* in rodents.

One might ask, in consideration of the differences of arrangement which I have described, and in particular of the inverse relations which exist between the grey matter and the white, whether the two portions of the gustatory nucleus described above do not in reality constitute two distinct nuclei, having different functions. But there exist very serious arguments against this interpretation. Independently of the morphological continuity which is observed between these two formations, one must take into account the identical structure of the grey matter in the two portions. We shall subsequently see in fact that the form of the myeline network and of the cells does not change throughout the whole extent of the nucleus in question. In addition, we find the presence of degenerated fibres, not numerous, it is true, but very distinct, in points which already belong to the inferior portion of the nucleus. So, in my opinion, we must conclude that the gelatinous substance of the solitary bundle is nothing but the inferior portion of the gustatory nucleus.

With regard to the structure of the gustatory nucleus, I shall merely indicate here the most elementary points. Its myelinated fibres are very fine and slightly curled, both in the white matter and in the grey. This results in the formation



of a felt-work which, seen under low power, has a somewhat blurred (*estompé*) appearance different from that of the surrounding grey mass. This network is slightly less fine in the inferior part of the nucleus than in the superior part.

The cells, studied in sections stained by carmine, are of a rounded or ovoid form; they measure from 15 to 20 mm. in diameter.

From the preceding observation it appears that one part of the presumed gustatory nucleus is reserved for the termination of the fibres of the fifth pair; consequently it may be assumed that the trigeminus is in itself a gustatory nerve, without necessarily implying that the sensory functions of the lingual are related exclusively to the chorda tympani which joins it. The gustatory nerve then, as a whole, seems to be formed by three branches leading to the same bulbo-pontine nucleus, one of which is attached to the fifth pair, another to the seventh, and a third to the ninth. Amongst the gustatory fibres of this last pair, some follow an ascending course (cerebral prolongation of the solitary bundle), whilst others blend with the solitary bundle proper in order to pass into the gelatinous substance of this bundle. It is extremely likely that the solitary bundle does not contain gustatory fibres below the point at which its gelatinous substance terminates, that is to say below the first 3 or 4 millimetres of its course.

I shall not here enter into the discussion of contradictory opinions of physiologists; I shall confine myself to comparing with the case which I have just reported a remarkable anatomoclinical observation which gives direct evidence of the gustatory properties of the trigeminus, and which consequently furnishes the complement of the demonstration which I have attempted.

This observation was reported by Wallenberg. It related to a patient who had presented anæsthesia in the region of the left trigeminus along with disappearance of taste on the back of the left tongue and a left hemiatrophy of the tongue. Autopsy showed the existence of a tumour affecting the hypoglossal, and partially the trigeminus; the glosso-pharyngeal and the intermediary nerve of Wrisberg were spared. From this fact Wallenberg concluded that taste fibres pass into the trigeminus. The investigation was very completely carried out by Marchi's method. At the level of the knee of the facial, there is detached

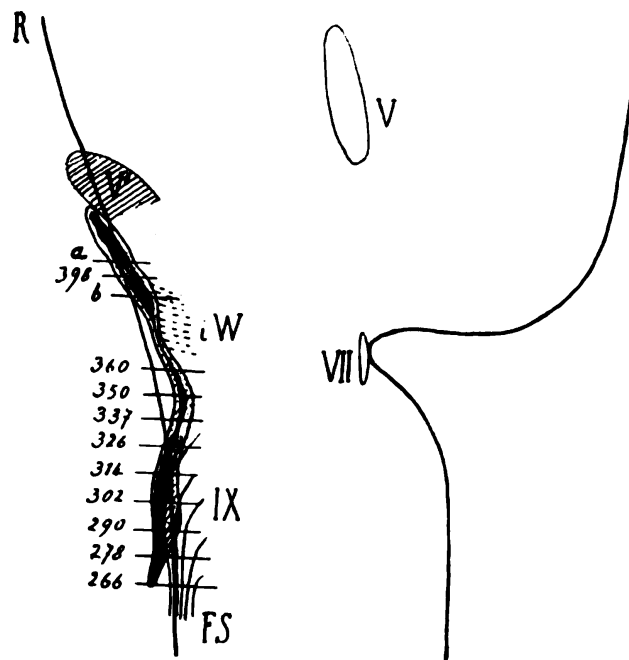


FIG. 7.

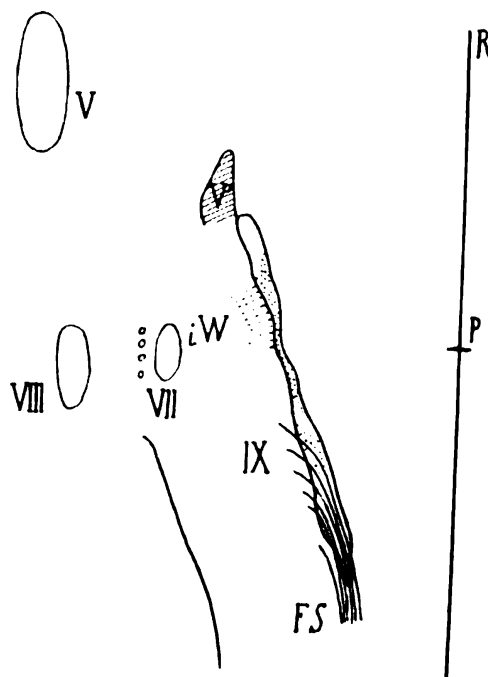


FIG. 8.



from the dorsal angle of the descending root of the trigeminus an oval piece of gelatinous substance, surrounded by degenerated fibres, which turn inwards and backwards. After the appearance of the internal nucleus of the acoustic, it takes its place at its ventral angle and goes directly into the nucleus of the solitary bundle. The number of degenerated fibres, at first much greater than that of the healthy fibres, gradually diminishes ; before the entry of the glosso-pharyngeal, it has fallen to its minimum and is limited to some elements which terminate, some at the ventral margin of the gelatinous substance of the solitary bundle, the others, after having skirted the solitary bundle, in its dorso-median portion. Throughout the whole bulbar course of the solitary bundle, as far as the interior angle of the calamus, there is a slender group of degenerated longitudinal fibres, which unite the posterior angle of the root of the trigeminus with the antero-internal margin of the solitary bundle. At the inferior extremity of the olive these fibres disappear, and there remains in this region only the descending root of the trigeminus, which goes down as far as the second cervical.

The oval portion of gelatinous substance surrounded by degenerated fibres which, to use Wallenberg's expression, detaches itself from the dorsal angle of the descending root of the trigeminus, is very evidently the superior part of the nucleus described above under the name of gustatory nucleus, the inferior part of this grey formation, which goes to join the solitary bundle, being simply the portion of this nucleus represented in fig. 8 (sections 360, 350, 337, and 326). My observation thus entirely accords with that of Wallenberg, and comparison between the two observations, one of which completes the other, strengthens the conviction that we are here dealing with a nucleus whose function is the reception of gustatory sensations in the bulbo-pontine region. We may also, from comparison of the two cases, draw this deduction, that the nucleus in question is divided into two territories, the inferior of which is reserved more particularly for the glosso-pharyngeal, whilst the superior is common to the trigeminus and the intermediary ; in the margin which separates these two territories, however, there exists a mixed zone in which the fibres from the three gustatory nerves blend with each other.

*History.*—Such are the conclusions to which we are led by study of pathological preparations in man, by the aid of Marchi's method. It is well to compare these results with those at which anatomists and experimental physiologists have arrived.

Anatomical study of the bulbo-pontine course of the intermediary nerve of Wrisberg was commenced in 1880 by Mathias Duval, by the aid of serial sections, stained in carmin and blue soluble in alcohol, from normal portions of the Cebus ape and of man. M. Duval has traced the root fibres of this nerve across the bulbar root of the trigeminus and into "a small, grey, ovular nucleus formed by cells of medium dimensions, and which is nothing but the continuation of the grey sensory column of the glosso-pharyngeal." From that connection M. Duval draws the anatomical conclusion that "the intermediary nerve of Wrisberg is one of the fascicles of the glosso-pharyngeal." M. Duval's anatomical description is remarkably exact, but it is incomplete, for the learned French histologist has not followed the nucleus in question above the point at which it receives fibres from the intermediary. Further, we have seen above that it is not the grey sensory column of the glosso-pharyngeal which constitutes the inferior extremity of the nucleus of the intermediary, but a special, very limited nucleus (gelatinous substance of the solitary bundle) which is situated round the highest portion of the solitary bundle, and in which only a small part of the fibres of this bundle terminate. M. Duval's description is accepted by Kölliker in these essential points, with the reserve which I have just indicated with regard to the gelatinous substance of the solitary bundle.

Roller, soon after, described under the name of the *radix descendens* of the gustatory nerve, the cerebral prolongation of the solitary bundle; according to this writer there are at this point fascicles which apparently unite with the spinal root of the trigeminus, and which penetrate into the convolutio trigemini.

His, in 1890, while studying the ganglion of the facial in a human embryo, found that its central prolongation joins the glosso-pharyngeal in the solitary bundle.

Martin, in embryos of the cat, has traced a bundle which ascends from the ninth pair to the fifth.

Cramer has described in a more precise way the formation to which Roller gave the name of descending root of the gustatory

nerve; he has seen a prolongation of the solitary bundle, with its grey nucleus, which was directed upwards from the point of the glosso-pharyngeal reflection. The grey matter in question approached nearer and nearer to the root of the trigeminal, and ended by blending with the gelatinous substance which accompanies this root. In a word, Cramer distinctly saw the inferior portion of the gustatory nucleus, but he did not distinguish its superior portion. Wallenberg is the only writer who, to my knowledge, has seen this superior portion before me.

Dexter has shown in embryos of the rabbit that the fibres of the solitary bundle come from the trigeminal, from the acoustico-facial, from the glosso-pharyngeal, and from the pneumogastric nerves.

In 1900 Van Gehuchten studied the course of cranial nerves by the experimental method; he pulled out the facial nerve in rabbits, and showed, by Marchi's method, the course of the fibres of the intermediary. These descend, occupying the apex of the postero-internal extremity of the trigeminus; then they penetrate into the solitary bundle, take a place inside the fibres of the glosso-pharyngeal, and descend to below the point of entry of the pneumo-gastric. At the level of the point at which the fibres of the intermediary commence to turn slightly from the trigeminus, there appears a small bright nucleus, which is situated immediately in front of and slightly inside the degenerated fibres; this nucleus is directed lower down into an internal portion, which is continuous with the dorsal nucleus or the motor small-celled nucleus of the glosso-pharyngeal, and into an external portion which is simply the terminal nucleus of the fibres of the nerve of Wrisberg, and of the glosso-pharyngeal nerve.

Ramon y Cajal, who employed the method of Golgi, describes in the same way the course of the intermediary. He draws attention to the fact that these fibres do not present a terminal bifurcation any more than do those of other nerves which enter into the composition of the solitary bundle; they are furnished only with collaterals. This observation agrees well with that of Van Gehuchten, who has not seen the ascending portion of the intermediary, but merely a compact group of fascicles which curve downwards in order to join the solitary bundle.

The distribution is different, at least apparently, in the case which I have studied, since there very distinctly exists an ascend-

ing portion of the intermediary nerve, the fibres of which ascend into the nucleus above the point of penetration of the root filaments. And further, the existence in adult man of an ascending portion of the solitary bundle, noted by several writers, seems also in contradiction to the results of investigation in animal embryos by Golgi's method. All the histologists who have employed this method agree in fact in denying the existence of a terminal bifurcation of roots destined for this bundle. These fibres simply curve downwards as if they represented only the inferior branch of the spinal roots, no superior branch existing. Does the distribution of the fibres in question differ in adult man from that in the embryo of mammiferous animals? Is it a question of age or of species? Or is the contradiction merely an apparent one, resulting only from a detail of arrangement of the fibres? I cannot answer this at present, but the point appears to me an interesting one to study.

Finally, I would draw attention to the fact that in man the fibres of the intermediary descend much less far than in the rabbit, and that from this fact there would seem to be an ascent of the gustatory nucleus in the human race.

#### DESCRIPTION OF FIGURES.

Fig. 1.—Degeneration of the intermediary nerve of Wrisberg; view of its whole extent; Marchi's method. Section corresponding to line 6 of fig. 7. The intra-pontine portion of the intermediary nerve alone is degenerated; the peripheral portion appears healthy. Retrograde degeneration of the facial nerve, which is the more marked the nearer it approaches to the nucleus of origin.

D, nucleus of Deiters; i.w., intermediary nerve of Wrisberg; n.g., gustatory nucleus; o.s., superior olive; P., pyramidal fascicle; v., vii., viii., trigeminal, facial, acoustic.

Fig. 2.—Gustatory nucleus above the point of penetration of fibres from the intermediary. Section corresponding to line a in Fig. 7. Degenerated fibres passing from the intermediary into the grey and into the white matter of the nucleus, throughout the whole of its internal aspect (ascending fibres of the intermediary).

S.c.i., internal semi-circular fibres.

Fig. 3.—Gustatory nucleus at level of point of penetration of the fascicles of the nerve of Wrisberg (enlarged portion of fig. 1).

T, corpus trapezoideum.

Fig. 4.—Gustatory nucleus below the point of penetration of the fascicles of the nerve of Wrisberg. Degenerated fibres in its grey and in its white matter.

P.c.i., inferior cerebellar peduncle.

Fig. 5.—From normal brain. Weigert-Pal method. Section following line 398 of fig. 7.

The gustatory nucleus forms four small rounded grey masses of matter, enclosed by a crown of white matter; in front are seen the nucleus and the descending root of the trigeminus; behind the internal semi-circular fibres of the cerebellum; to the right of the figure the facial nerve, to the left the corpus trapezoideum.

Fig. 6.—From normal brain. Weigert-Pal method. Inferior portion of the gustatory nucleus studied by a series of sections one-twelfth mm. in thickness, about 1 mm. distant from each other. Section 266: termination of the gustatory nucleus behind the solitary bundle. Sections 266 to 326: successive forms of the portion of the gustatory nucleus which is in connection with the solitary bundle (gelatinous substance of the solitary nucleus). Sections 337 and 350: cerebral prolongation of the solitary bundle (ascending fibres of the glosso-pharyngeal). Section 360: the gustatory nucleus has become connected with the trigeminal, and has assumed the aspect which it retains through the whole extent of its superior portion.

Fig. 7.—Projection of the gustatory nucleus on an antero-posterior plane (enlargement of 3 diameters). The dotted line represents the area of distribution of the intermediary nerve of Wrisberg; the grey matter is represented in black.

Fig. 8.—Projection of the gustatory nucleus on a vertical and transverse plane.

#### BIBLIOGRAPHY.

1. Alexander Bruce. "On the Dorsal or so-called Sensory Nucleus of the Glosso-pharyngeal Nerve, and on the Nuclei of Origin of the Trigeminal Nerve," *Brain*, xxi., 1898.
2. Alexander Bruce. "Illustrations of the Mid and Hind Brain," 1892.
3. Cramer. "Beitrag zur feineren Anatomie der Medulla oblongata und der Brücke." Fischer, Jena, 1894.
4. Dexter. "Ein Beitrag zur Morphologie des verlängerten Markes beim Kaninchen," *Arch. f. Anat. u. Physiol.* (anat. Abth.), 1895.
5. M. Duval. "Recherches sur l'origine réelle des nerfs craniens," *Journ. de l'Anat.*, 1880.
6. Van Gehuchten. "Recherches sur la terminaison centrale des nerfs sensibles périphériques," *Le Névrase*, i., 1900.
7. Kölliker. "Handbuch der Gewebelehre des Menschen," 6th ed., t. ii.
8. Nageotte. "Sur la nature et la pathogénie des lésions radiculaires de la moelle qui accompagnent les tumeurs cérébrales," *Rev. Neurol.*, 1904.



9. Roller. "Der centrale Verlauf des Nervus Glossopharyngeus," *Arch. f. mikros. Anat.*, xix., 1881.
10. Ramon y Cajal. "Textura del sistema nervioso del hombre y de los vertebratos," t. ii.
11. Sapolini. "Étude anatomique sur le nerf de Wrisberg et la corde du tympan," *Journ. de méd. de Bruxelles*, 1884.
12. Wallenberg. "Das dorsale Gebiet der spinalen Trigeminiwurzel und seine Beziehungen zum solitären Bündel beim Menschen," *Zeitschr. f. Nervenheilk.*, xi., 1897.

### **THE DESCENDING DEGENERATIONS OF THE POSTERIOR COLUMNS IN (1) TRANSVERSE MYELITIS AND (2) AFTER COMPRESSION OF THE DORSAL POSTERIOR ROOTS BY TUMOURS.**

By DAVID ORR, M.D.,  
County Asylum Prestwich, Manchester.

THE posterior columns of the cord contain, besides ascending tracts, descending fibres traversing the areas known as Schultze's comma, Hoche's marginal zone, Flechsig's oval, and the triangle of Gombault and Philippe. These are now admitted by the majority of writers to be portions of one continuous tract (Barbacci, Déjerine and Theohari, Stewart, Flatau), the last three lying in the postero-median part of the columns, while the first, pushed outwards in all probability by the gradual formation of Goll's fasciculus, comes in the cervical and dorsal regions to lie alongside the posterior horn.

Opinion regarding the position of these tracts is unanimous; but regarding the origin of the fibres composing them, especially the comma tract, the opposite holds. Some have definitely asserted that in the comma there are only fibres of exogenous origin, the descending branches of the posterior roots; others place their seat of origin in the cord exclusively; others again think the tract contains fibres derived from both sources.

With a view to determining where the fibres originate, cases of cord compression, myelitis, compression of the spinal roots by tumours or meningitis, have been studied. The posterior roots have also been divided in animals and the cord examined for degeneration in Schultze's comma, with negative (Tooth) and

positive results (Oddi and Rossi, Löwenthal, Flatau, Van Gehuchten, Margulies).

According to Van Gehuchten the descending branches of a posterior root form a scattered bundle, at first in the immediate neighbourhood of the posterior horn, but afterwards lying more internally. After degeneration, the resulting sclerosis is hardly appreciable. The fibres are short, and never extend downwards for more than six or seven segments.

In Flatau's experiments these descending branches lay at first in the root entry zone, but ultimately occupied the region around the anterior part of the median septum and behind the commissure.

In man also, after compression of the posterior roots, the comma tract shows descending degeneration for a few segments (Zappert, Schaffer, Jacobsohn, Homén, Laslett and Warrington), so that we have ample evidence of the presence of exogenous fibres in this zone.

But a different series of observations shows that the comma tract contains, in addition to the short exogenous fibres, other fibres of sufficient length to reach from the cervical region to the lowest sacral.

After transverse division of the cord by myelitis or injury, Schultze's comma is degenerated downwards as far as the lower dorsal region as a very definite bundle; and the degeneration is continued into the zones around the median septum, in the lumbar and sacral cord (Daxenberger, Hoche, Achalme and Theohari, Déjerine and Theohari, Stewart, Bruce and Muir).

In the descending tract as a whole there must therefore be long descending fibres of purely endogenous origin, which, judging from the degree of degeneration in Flechsig's oval in cases of cord compression, must in the comma greatly exceed in numbers the exogenous ones.

Of the endogenous nature of Flechsig's oval we have additional evidence from cases of *tabes dorsalis* in which this area is always found intact, even in the later stages of the disease (Bruce, Homén, Orr and Rows).

Recently I had the opportunity of examining two spinal cords, one from a case of Transverse Myelitis obtained through the kindness of Mr Platt, Surgeon to the Manchester Royal Infirmary, the other from a case of my own exhibiting Multiple Tumours com-

pressing the posterior spinal roots in the dorsal region. The interest of these two cases consists in the different degree of degeneration exhibited in the descending tracts of the posterior columns in a *myelitic* or *endogenous* lesion on the one hand, and by a *root* or *exogenous* lesion on the other. It will be seen that in the latter instance, although such a high degree of root destruction was present, yet Hoche's, Flechsig's, and Gombault and Philippe's zones showed a very slight degeneration. Even this slight degeneration I am not prepared to accept as of the descending fibres of posterior roots, for in the lower dorsal region there was a small tumour of the posterior columns growing inwards from the cord margin, and in addition there were several myelitic foci on either side of the median septum. Bearing in mind that the endogenous fibres of Schultze's comma pass backwards at this level to gain the septo-marginal region, one is confronted with the probability that some of these fibres were implicated in their course.

CASE I.—The patient from whom this cord was obtained developed transverse myelitis on July 26th, 1905, and died September 12th, 1905.

Marchi's method alone was used to trace the degenerations. The entire cord, medulla, and pons were examined. The myelitic focus was found to affect the 8th dorsal and the upper part of the ninth dorsal segments. The degeneration was most marked in the posterior columns, the postero-lateral region, and the anterior radicular zones of the cord. In the grey matter, and regions occupied by the basis bundles, there was considerable degeneration, but this was less in comparison with that in the areas just mentioned.

As the interest in the two cases to be described centres entirely in the descending degenerations observed in the posterior columns of the lumbo-sacral region, I do not propose to give the ascending ones in detail.

Both cerebellar tracts and Goll's column were completely degenerated. The path pursued by the fibres in the former was that usually given, viz., the postero-lateral, through the inferior peduncle of the cerebellum, the antero-lateral, by the superior peduncle. In Goll's column the fibres were traced into the gracilis nucleus, where the great majority ended; but not all, as a by no means inconspicuous number could be seen to cross by

the arcuate fibres to the fillet. This confirms the observation already made by Stewart (*Brain*, Summer, 1901).

Finally, degeneration was present in a tract occupying the anterior sulco-marginal region, and was followed into the medulla. Here, having lost many fibres, it seemed to end posterior to the inferior olive, and somewhat towards its inner extremity.

Below the myelitis there was descending degeneration. The crossed pyramidal tracts could be recognised in the lowest segment of the cord, the direct as far as S<sup>3</sup>. There was a very distinct degeneration in the sulco-marginal region as far as S<sup>2</sup>; and in the antero-lateral portion of the cord in front of the crossed pyramidal tract there were many scattered degenerated fibres.

#### *Descending Degenerations of the Posterior Columns.*

In the centre of the myelitic focus the whole posterior column was uniformly filled with Marchi reaction, but a short distance below this, in the lower part of D<sup>9</sup>, a rapid diminution in the extent of the degeneration occurred. The external portion of each column still showed a dense mass of degenerated fibres, which at this level took definite shape only in the cornu-commissural angle. In D<sup>10</sup>, however, where the central portions of the columns showed a very few degenerated fibres, there was a definite bundle on each side extending from the commissure backwards to the cord periphery and lying somewhat retracted from the posterior horn. It will be seen in photo 1 how this bundle broadens posteriorly, where the fibres are more scattered. Throughout D<sup>11</sup> and D<sup>12</sup> the posterior part of this tract (Shultze's comma) gradually became more indefinite and receded a little from the cord margin; and, in the latter segment, Hoche's marginal tract first made its appearance as a small band consisting of a few fibres alongside the posterior extremity of the median septum.

In L<sup>1</sup> and L<sup>2</sup> the tail of the comma tract gradually disappeared, the head became ill-defined, and the degenerated fibres became scattered diffusely in the more central portion of each column. In L<sup>3</sup> there was no trace of the comma tract, but the septo-marginal bundle was well defined. The fibres of this tract lay as a thin band, situated for a short distance along the cord

margin on either side of the median septum, and spreading forward on either side of its posterior extremity. In L<sup>4</sup> there were many scattered fibres in the posterior columns. These were more abundant in the central parts, and also around the anterior part of the median septum, as if the head of the comma had become diffused there. The number of degenerated fibres in the septo-marginal bundle had increased, and the tract extended further in a ventral direction (photo 2).

In L<sup>5</sup> (photo 3) one sees how the septo-marginal tract gradually becomes the central oval of Flechsig. A few degenerated fibres are still present at the cord margin behind the oval. In front of Flechsig's area there were many degenerated fibres lying close to the median septum, and extending forwards to the posterior commissure, where, spreading, they formed a figure of a somewhat triangular shape, whose apex pointed backwards.

Passing downwards through S<sup>1</sup> and S<sup>2</sup> (photos 4 and 5), one found Flechsig's oval field enlarged by the degenerated fibres at the cord margin and those behind the commissure passing respectively forwards and backwards.

Below S<sup>2</sup> there was a malformation of the cord which prevented the descending fibres from taking up the position which they ought to do normally. A mass of grey and white matter, appearing first just behind the central canal, grew, in the middle line, downwards into the posterior *columns*, separating and rotating them outwards. In this malformation were developed two rudimentary anterior and posterior horns, until finally the cord became double, but only for a short distance. The malformation was continued to the end of the cord as a partial doubling. In consequence, the triangle of Gombault and Philippe was divided into two portions, each widely separated from the other. In these, degenerated fibres were recognisable to the cord extremity (photo 6).

In Case II. there were Endotheliomata scattered throughout the central nervous system, one of which was situated in the right frontal lobe, another involved the crura cerebri and third nerves, while on the spinal roots of the dorsal cord there were many (see *infra*).

The spinal root lesions are alone of interest at present.

For the examination of the lesions, Marchi's, Van Gieson's, and the hæmatoxylin and eosin methods were used.

The tumours of the dorsal cord, situated on both anterior and posterior nerves, originated in connection with the perineural sheath, and varied considerably in size; some were round, others fusiform. The majority totally or almost totally destroyed the nerves. Others again—though rarely—affected a few fasciculi only. As a result, one found a cellulifugal degeneration; and where the sensory roots were involved, a degeneration of the posterior columns.

In the cervical cord only two, the second and fifth left posterior roots, were affected; only part of each root had undergone degeneration, so that the root entry zone was only partially filled with degenerated fibres.

In the dorsal cord the appearances were very different, for here in every segment from D<sup>2</sup> to D<sup>12</sup> the root entry zone was filled with degenerated fibres, the result of destruction of the posterior roots.

Both of these were destroyed in all segments with the following exceptions: D<sup>2</sup>, in which only the right posterior root was implicated; D<sup>12</sup>, only the left.

The anterior roots affected were: in D<sup>3</sup> and D<sup>4</sup>, the left; in D<sup>7</sup>, the right one; in D<sup>10</sup> and D<sup>11</sup>, the left; and in these last two the growth extended into the anterior radicular zone.

In D<sup>3</sup> and D<sup>4</sup> the tumours were large, and had invaded the lateral portion of the cord from the anterior radicular zone backwards almost to the posterior horn.

In D<sup>7</sup> a similar condition was present, but the invasion of the lateral part of the cord was less in depth, and was confined more to the postero-lateral region.

The degeneration of the posterior columns resulting from such an extensive root destruction attained very considerable proportions, and was followed up to the medulla, where the majority of the degenerated fibres ended in the nucleus gracilis; many, however, passed into the nucleus cuneatus.

As in Case I., some of the fibres passed through these nuclei and reached the fillet by the arcuate fibres; others passed on into the anterior external arcuate bundle, and were traced round the ventral aspect of the pyramids to end finally in the restiform body.

The invasion of the lateral surface by the growth in D<sup>3</sup>, D<sup>4</sup>, and D<sup>7</sup>, caused an ascending degeneration of the cerebellar

tracts. Both were completely destroyed on the left side; but only the direct postero-lateral and the posterior part of the antero-lateral (Gower's) on the right.

In D<sup>11</sup> there was a tumour in the posterior columns. This was small, with its base at the cord margin on either side of the median septum, and spread ventrally along it for a short distance. From this point upwards there was a continuous ascending degeneration as far as the medulla.

Regarding the descending degeneration of the pyramidal paths, following implication of the crura, it is only necessary to state that this was more marked in the left C. P. T. and in the right D. P. T., and was followed to the same levels as in Case I.

Throughout the cord there was considerable general sclerosis and vascular degeneration. The sclerosis was more accentuated, however, in the degenerated tracts—especially in the posterior columns—and around the cord margin. The vessels and pia-arachnoid showed a high degree of hyaline thickening, and the latter in many places sarcomatous infiltration, especially in the neighbourhood of the tumours.

#### *Descending Degenerations of the Posterior Columns.*

As already noted, the root entry zones in the dorsal cord were filled with degeneration from D<sup>2</sup> to D<sup>12</sup> (photo 7), but, in addition, there were lesions in the lower segments of equal importance.

In D<sup>11</sup> there was the small tumour already noted at the posterior extremity of the median septum. This involved not more than one-eighth of an inch of the segment, in a vertical direction. In D<sup>7</sup>, D<sup>8</sup>, D<sup>9</sup>, D<sup>10</sup>, and D<sup>11</sup> there was considerable degeneration and sclerosis in the region of the postero-lateral septum as well as round the margin of the cord (photo 7).

In D<sup>7</sup> there was a small myelitic softening situated at the cord margin just internal to the left posterior root, in D<sup>8</sup> one on the right side of the hinder third of the median septum, and several small areas of sclerosis in each column close to the periphery (photo 8).

In D<sup>12</sup> there were two degenerated and sclerosed patches, one on either side of the posterior extremity of the median

septum. The margin of the right column was almost entirely free from degenerated fibres. There was still some degeneration with advanced sclerosis around the left postero-lateral septum (photo 9). In the right root and entry zone there was little degeneration in comparison with that in the left root, and in those of the segments immediately above.

On comparing this section with one taken one segment below the myelitis in Case I., the difference was very marked. Here there were only a few scattered fibres in the position of Schultze's comma tract, thus forming a striking contrast with the well-defined bundle in Case I. (photo 1).

In the 1st lumbar segment (photo 10) the degeneration in the root entry zones was much less than in D<sup>12</sup>, especially on the right side, and was continuous with that of the entering root.

There was no trace of a comma, and the posterior part of the columns was free from degeneration with the exception of a few fibres in the position of Hoche's marginal zone on the left side. In all the lumbar segments the posterior roots and entry zones showed a slight degree of degeneration, which gradually diminished from above downwards. In L<sup>2</sup> there was a small sclerotic patch on the right side of the posterior extremity of the median septum, while on its left side the septo-marginal tract first made its appearance as a definite structure.

In L<sup>3</sup> and L<sup>4</sup> the septo-marginal tract left the periphery, and in L<sup>5</sup> became Flechsig's oval. The oval in this case contained far fewer degenerated fibres than that of Case I., as a comparison of the photographs from each will show (photos 11, 3, 4); and lying along the median septum anteriorly there were only a few degenerated fibres, scattered too irregularly to form a definite band. This band, stretching forwards to the commissure, was very definite in Case I.

In the sacral cord the degenerated fibres of this descending tract rapidly diminished in numbers, but a few could be recognised in the triangle of Gombault and Philippe, even as far as the lowest sacral segment.

*To summarise* the descending lesions in the two cases, we find in the first many degenerated fibres in Schultze's comma, which, becoming diffused about the upper lumbar region, reappear in very definite tract form in the dorso-median bundles below. In the second, immediately the gross lesions of the roots ceased,



on the right side at  $D^{11}$ , and on the left at  $D^{12}$ ; the root entry zones contained only a scanty and scattered degeneration. No comma tract existed in the right entry zone of  $D^{12}$ , nor in either at  $L^1$ , whereas in  $D^{12}$  of Case I. Schultze's comma was a prominent structure, especially in its ventral portion.

If, then, in an extensive posterior root lesion, Schultze's comma fails to show degeneration, while this is so obvious at the same level after a myelitis, then we must conclude that by far the greater number of the fibres of the tract in question are endogenous.

Extending the same line of argument to the degenerations in Flechsig's oval, we have confirmatory evidence of the great preponderance of endogenous fibres in the descending tracts, for numerically the degenerated fibres in Case I. greatly exceeded those in Case II.

But the presence of the tumour and myelitic softenings in the lower dorsal region of the latter case renders it extremely doubtful if the slight degeneration in Flechsig's oval was exogenous. It is much more probable that it consisted of endogenous fibres intercepted, in the posterior regions of  $D^{11}$  and  $D^{12}$ , in their passage from Schultze's comma to the dorso-median zones. Naturally, with such lesions, the large majority would escape and continue their course without interruption, as these endogenous fibres traverse the columns in the transition from Schultze's comma to the septo-marginal zone and Flechsig's oval in a very diffused manner.

It appears to me, therefore, that Case II. demonstrates that the descending branches of the lower dorsal roots do not form a comma tract in the upper lumbar cord, nor enter Flechsig's oval, but pass almost at once into the grey matter.

#### REFERENCES.

1. Achalme and Theohari. (Cited by Flatau: "Handbuch der path. Anat. d. Nervensystems," 1904, vol. ii. p. 964.)
2. Barbacci. "Lo Sperimentale," 1891, p. 386.
3. Bruce and Muir. *Brain*, 1896, p. 333.
4. Bruce. *Brain*, 1897, p. 265.
5. Daxenberger. *Deutsche Zeitsch. f. Nervenheil.*, Bd. iv., 1893.
6. Déjerine and Theohari. *Journ. de Phys. et Path.*, March 1899.
7. Flatau. "Handbuch der path. Anat. d. Nervensystems," 1904, vol. ii.
8. Gombault et Philippe. *Arch. de Méd. Experiment*, 1894.

Head of  
Schultze's  
comma  
tract.

Tail  
ill-defined.

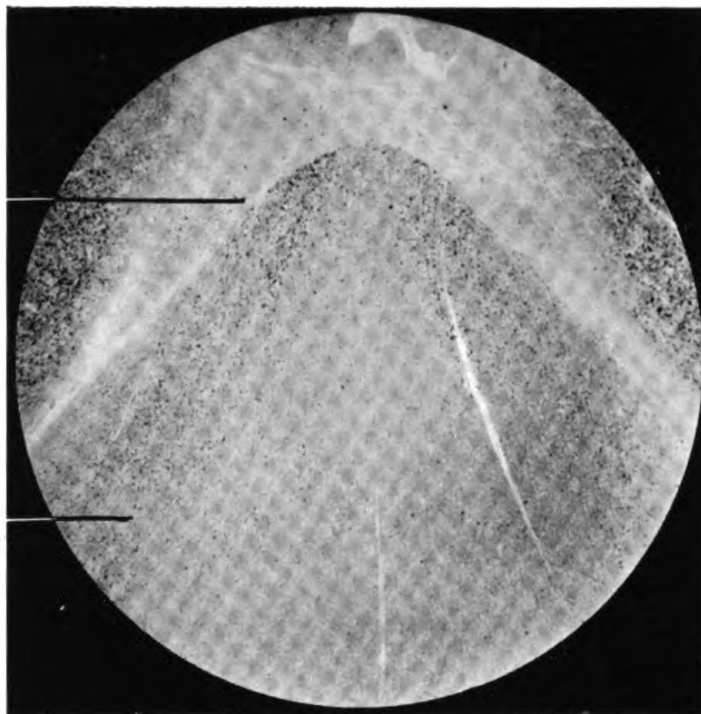


PHOTO 1.—Case 1. D<sup>10</sup>. Schultze's comma tract better defined anteriorly than in the posterior part of the columns.

Septo-  
marginal  
bundle.

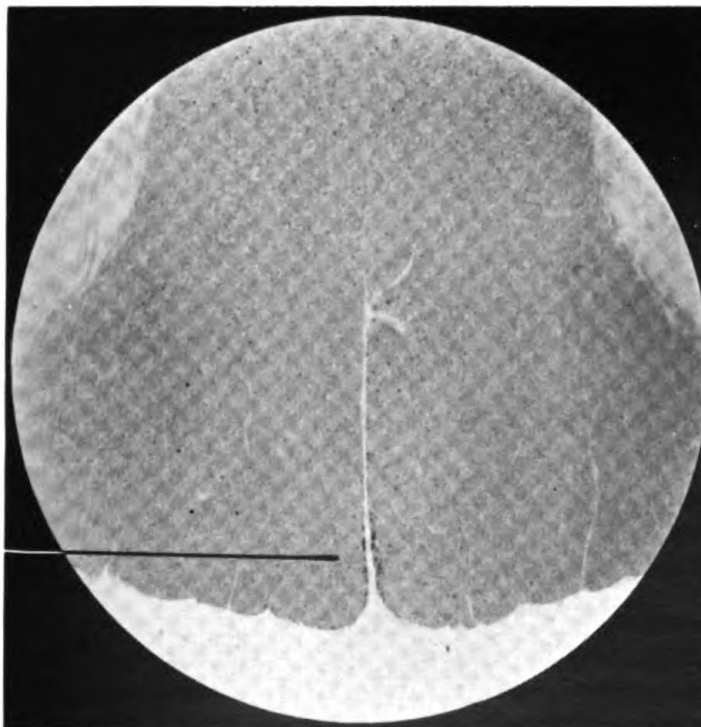


PHOTO 2.—Case 1. L<sup>4</sup>. Septo-marginal tract on either side of the postero-median fissure.



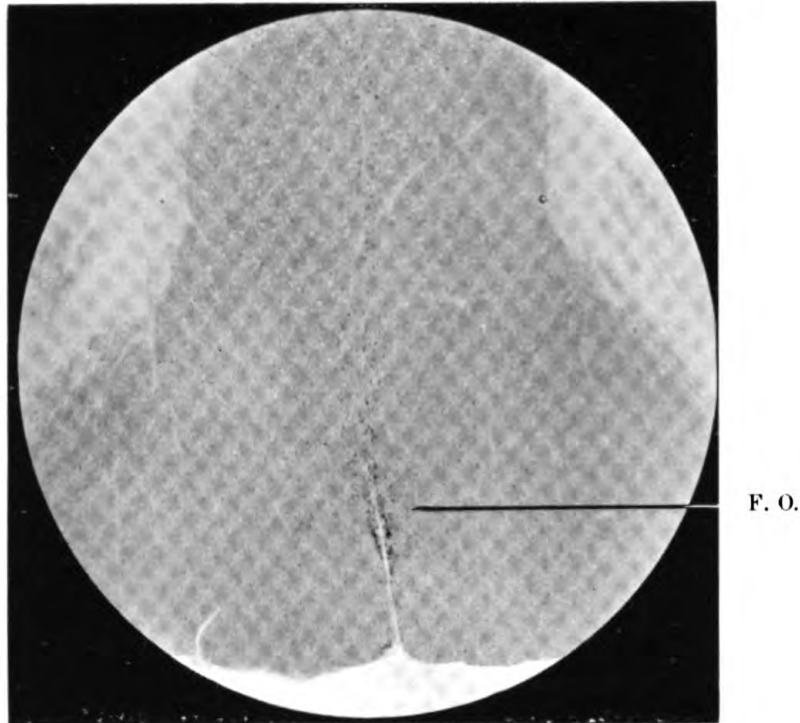


PHOTO 3.—Case 1. L<sup>5</sup>. Flechsig's central oval, just ventral to the posterior margin of the cord.

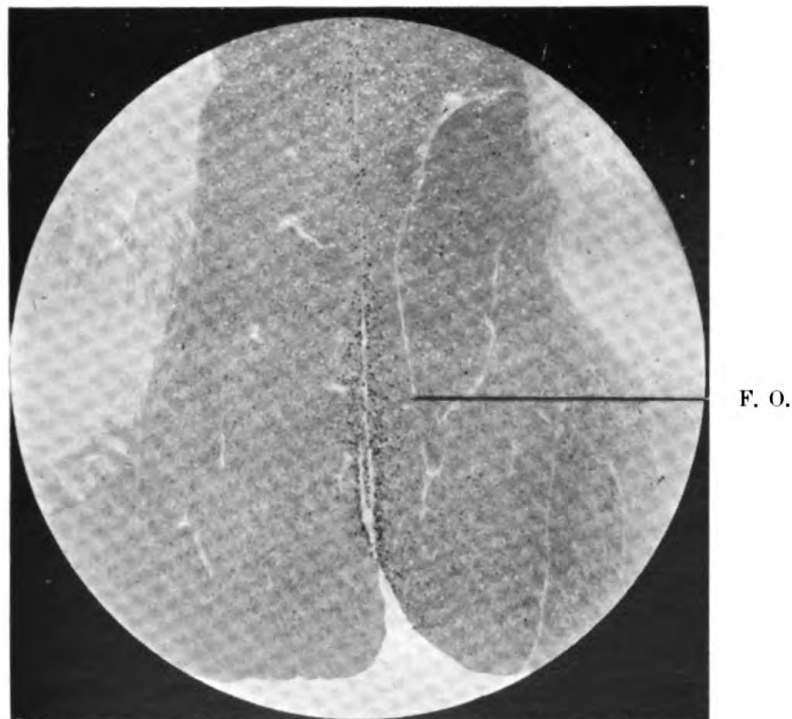
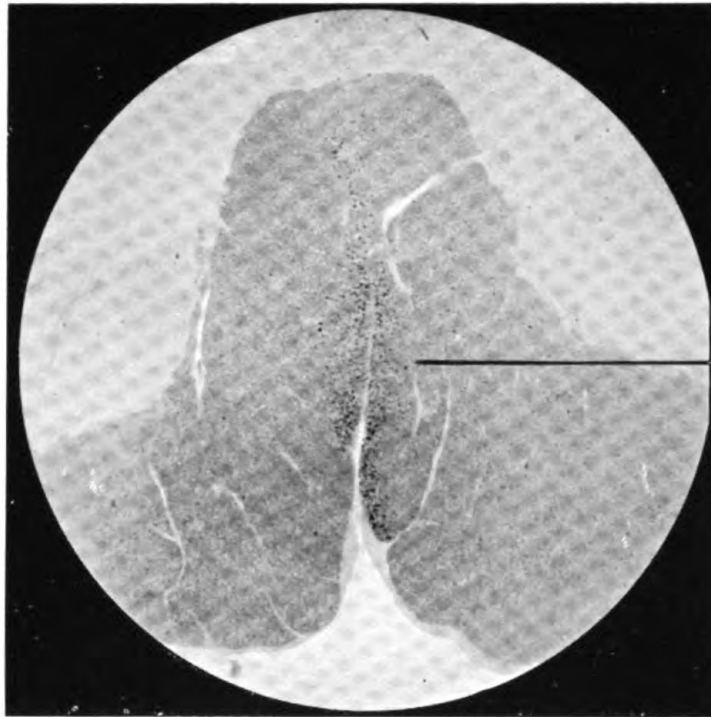


PHOTO 4.—Case 1. S<sup>1</sup>. Flechsig's central oval.





F. O.

PHOTO 5.—Case 1. S<sup>2</sup>. Flechsig's central oval.

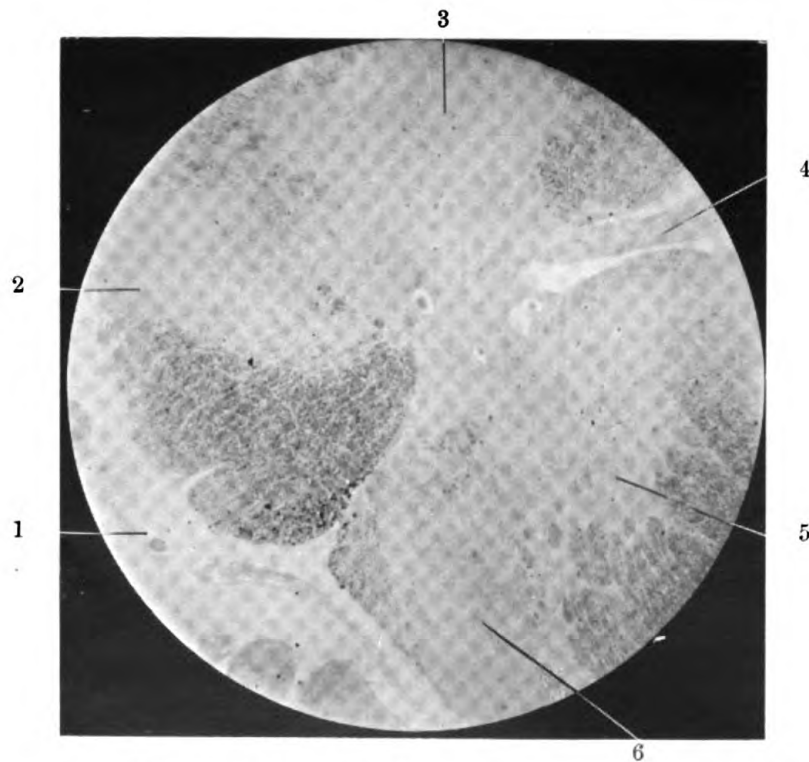


PHOTO 6.—Case 1. Cord almost completely double.

- |  |   |
|--|---|
| 1. Left half of triangle of Gombault and Philippe. | 4. Commissure.                              |
| 2. Original posterior horn.                        | 5. Rudimentary anterior horn. } New         |
| 3. Original anterior horn.                         | 6. Rudimentary posterior horn. } formation. |



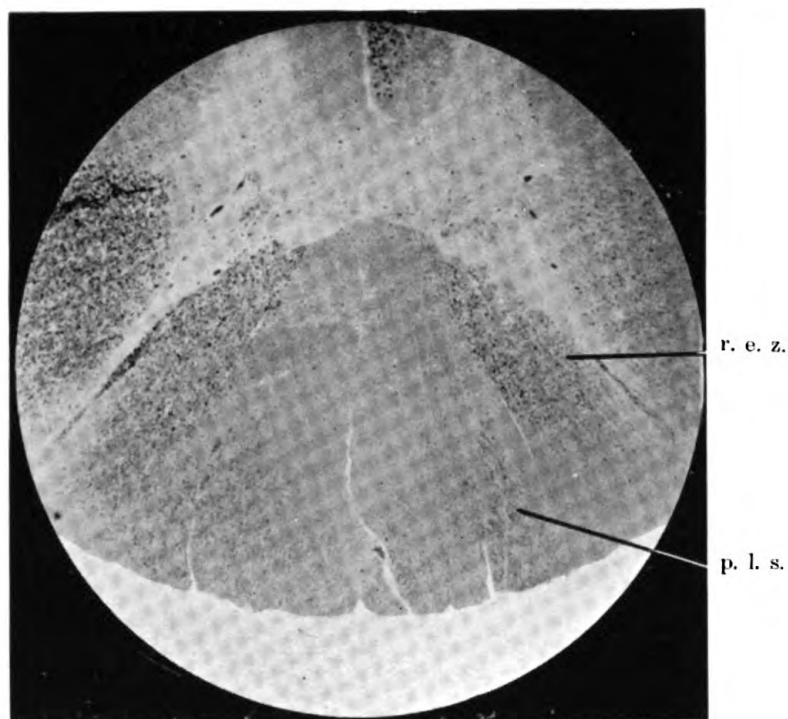


PHOTO 7.—Case 2. D<sup>10</sup>. Observe the degeneration (1) of the root entry zones on either side ; and (2) round the postero-lateral septum.

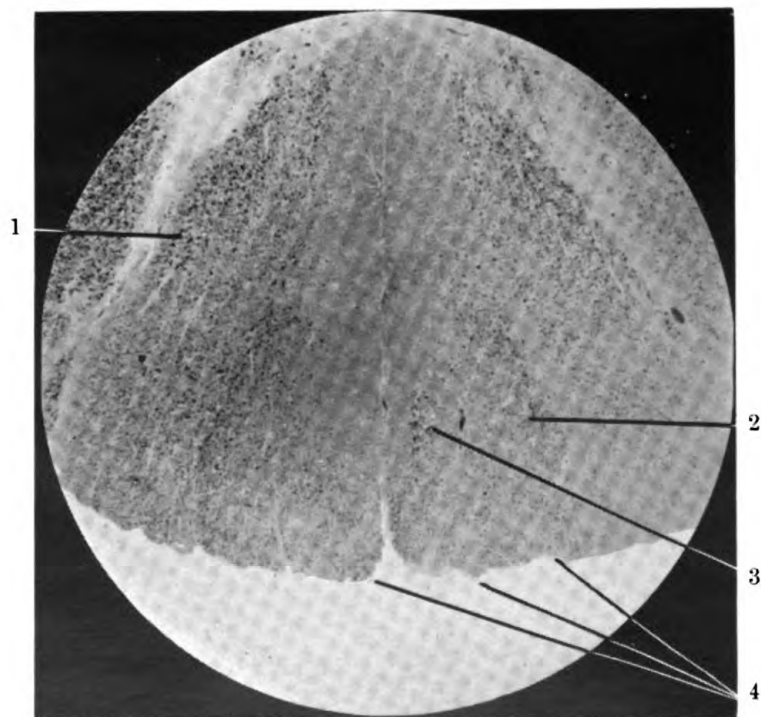


PHOTO 8 —Case 2. D<sup>8</sup>.  
 1. Degeneration in the root entry zones. 3. Small myelitic softening.  
 2. „ „ around postero-lateral septum. 4. Small areas of sclerosis.





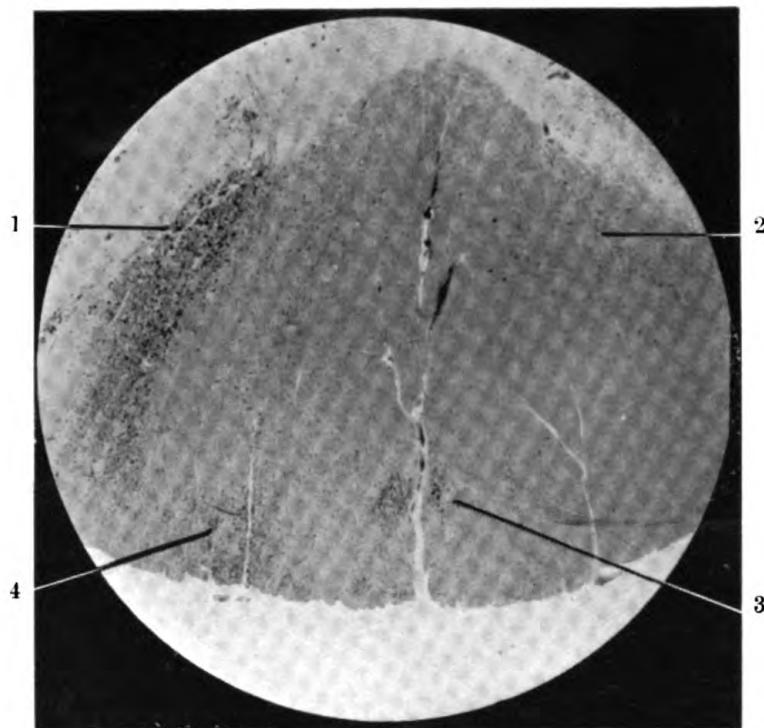


PHOTO 9. Case 2. D<sup>12</sup>.

1. Degeneration in left root entry zone.
2. A few degenerated fibres in right root entry zone.
3. Myelitic softening on each side of postero-median septum.
4. Degeneration around left postero-lateral septum.

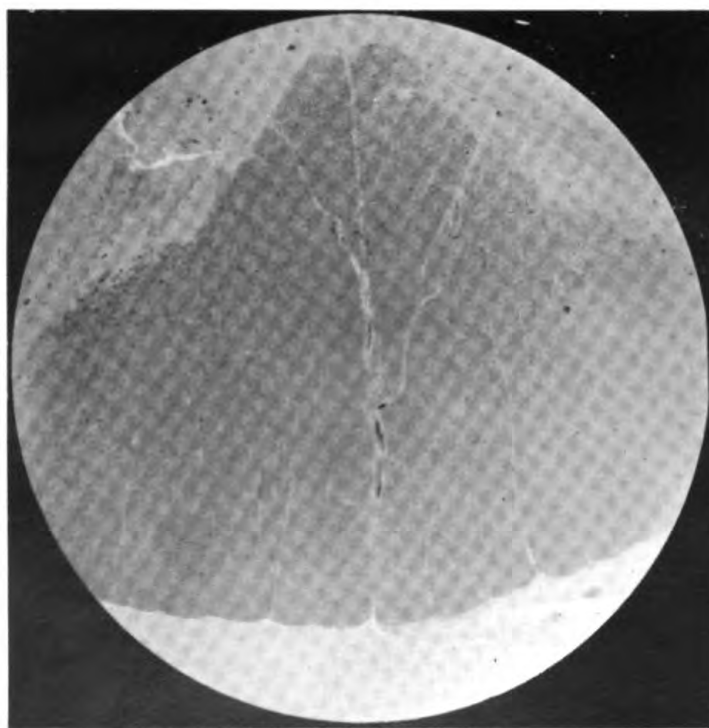


PHOTO 10.—Case 2. L<sup>1</sup>. Observe both root entry zones contain only a few scattered degenerated fibres.



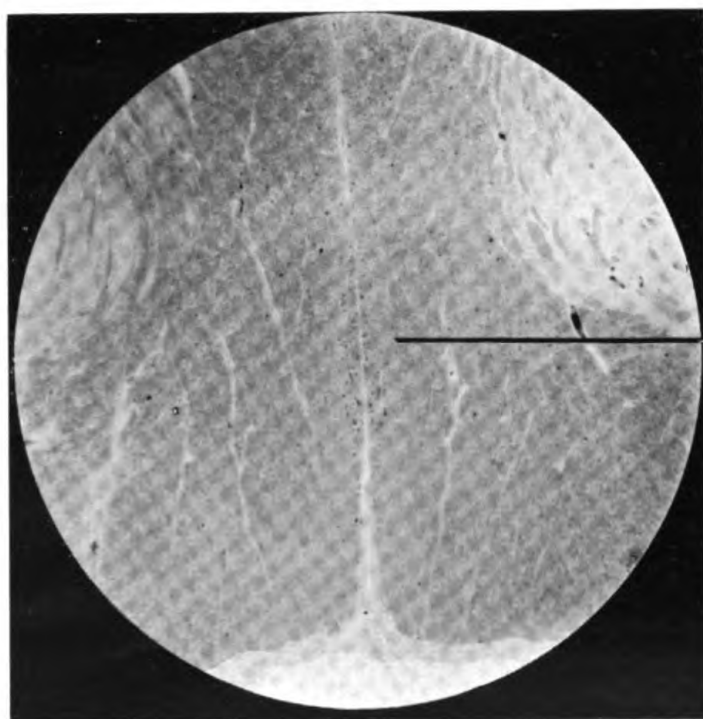


PHOTO 11.—Case 2. *15*. Note the small number of degenerated fibres in Flechsig's oval in this case.



9. Hoche. *Neurol. Centralb.*, 1896, p. 155.
10. Homén. *Deutsche Zeitsch. f. Nervenheil.*, p. 24, 1901.
11. Homén. "Handbuch der path. Anat. d. Nervensystems," by Flatau, etc., 1904, vol. ii.
12. Jacobsohn. *Zeitsch. f. klin. Med.*, Bd. xxxvii., 1899.
13. Laslett and Warrington. *Brain*, 1899.
14. Löwenthal. (Cited by Flatau. See above.)
15. May. *Rev. Neur. and Psych.*, No. 1, 1905.
16. Margulies. *Monatsch. f. Psych. u. Neur.*, 1897.
17. Oddi and Rossi. *Arch. Ital. de Biolog.*, 1891, p. 296.
18. Orr and Rows. *Brain*, winter, 1904.
19. Schaffer. *Arch. f. Mikroskop. Anat.*, 1894.
20. Stewart. *Brain*, summer, 1901.
21. Tooth. *Brit. Med. Journ.*, 1889.
22. Van Gehuchten. "Anat. du Système Nerveux de l'Homme," 1906.
23. Zappert. *Neurol. Centralb.*, No. 3, 1896.

## Abstracts

### PATHOLOGY

- A MALFORMATION OF THE SPINAL CORD APPARENTLY (268) HITHERTO UNDESCRIBED.** (Ueber eine bisher anscheinend nicht beschriebene Missbildung am Rückenmarke.) A. WESTPHAL, *Arch. f. Psychiat. u. Nervenkrankheiten*, Bd. 41, H. 2, S. 712.

THE cord was that of a woman of 30, who died with symptoms of dementia præcox of three months' duration. There were marked gluteal and trochanteric bedsores, giving rise to suspicions of some cord lesion.

The vertebral canal and dura were found normal, as was the cord down to the level of the upper lumbar region, but the lower lumbar cord appeared to divide into two parts; one, the left, larger than the other, but unconnected with any nerve roots. Serial sections showed first the appearance of unstriped muscle fibres in the posterior median fissure; these increased in amount downwards, being at first in continuity with the left posterior column, but soon becoming separate from the cord. At lower levels it was found that this left "cord" was an elongated cyst lined with cylindrical epithelium, its wall being formed of circular and longitudinal bundles of unstriped muscle with some fat, connective

tissue, and a few nerve fibres, but no nerve cells. The real cord contained numerous recent hæmorrhages, and a little way below the separation of the cyst showed a well-defined doubling. This began with the appearance of an independent second central canal on the inner side of one of the anterior horns, and caudalwards there gradually developed a third anterior and posterior horn, the former containing numerous motor cells. This condition persisted to the lower end of the cord.

The cyst was supposed to be a persistent part of the embryonic proctodeum or neurenteric canal. J. H. HARVEY PIRIE.

**ON THE ACTION OF VENOMS OF DIFFERENT SPECIES OF  
(269) POISONOUS SNAKES ON THE NERVOUS SYSTEM.**

**V. Venom of Common Krait (*Bungarus Coeruleus*).** GEORGE LAMB and WALTER K. HUNTER, *Lancet*, May 5, 1906, p. 1231.

THE common krait is a small snake, but its venom is of great toxicity, the minimum lethal dose being a third of that of cobra venom. But the symptoms following poisoning with either venom are almost identical, *i.e.* there is paralysis beginning in the skeletal muscles and gradually involving the muscles of respiration, death being due to failure of the respiratory mechanism.

The venom also acts on the circulatory apparatus much in the same way as does cobra venom, but krait venom acts in addition on the vaso-motor centre in the medulla, causing a well-marked fall of blood pressure, doubtless due to paralysis of the vaso-motor centre.

The authors have examined histologically the nervous systems of four monkeys dying at intervals of 16½, 12, 10, and 4½ hours respectively, after injection of this venom; also the tissues from a soldier who died a few hours after being bitten by a snake identified as a common krait. The first three monkeys showed well-marked degenerative changes in the motor ganglion cells throughout the whole central nervous system. In the fourth monkey (dead in 4½ hours) there were practically no chromatolytic changes to be found. With Donaggio's stain the peripheral nerves of the four monkeys gave undoubted signs of an early parenchymatous degeneration. In the human case, as with the fourth monkey, the histological examination of the central nervous system was negative; the peripheral nerves, however, were not examined.

Generally the degenerative changes produced by this venom are similar to those formerly described when dealing with the venoms of cobra and the banded krait (*Bungarus fasciatus*).

W. K. HUNTER.

## CLINICAL NEUROLOGY.

**ISOLATED TRAUMATIC PARALYSIS.** (Ueber isolierte traumatische Lähmung.) F. FISCHLER, *Neurolog. Centralbl.*, May 16, 1906, p. 444.

I. *Suprascapular Nerve*.—This occurrence is one of the rarities of neurology. The writer first describes a case which occurred in a woman as a result of the strain of stretching to reach a brush that was fixed outside a window. Immediate pain was felt in the right shoulder blade. The pain soon disappeared, and no other sensory symptoms were ever present. Weakness of the arm for outward movement, particularly in the raised position, was the only symptom. No atrophy or fibrillary tremors were seen, save in the supraspinatus muscle. The right shoulder was lower than the left, and the shoulder blade was displaced  $1\frac{1}{2}$  cm. outwards and downwards. External rotation of the humerus was impeded. The suprascapular nerve did not respond when tested electrically. The infraspinatus shewed diminished response to both faradism and galvanism; qualitative changes could not be established with certainty, nor could the reaction of the supraspinatus. Recovery ensued after sixteen weeks.

The diagnosis is discussed and the unusual etiology insisted on. The writer has found fourteen similar cases in the literature, seven of which were of traumatic origin; in three of these the trauma was direct, in four indirect.

II. *Right Musculo-cutaneous Nerve*.—The patient was a stoker, aged 30, who, two years before, had sustained an injury on the outer side of the right forearm in the lower third. The wound was sewn up, but suppurated, and healed only after five weeks. After this a pustular eruption appeared on the lower part of the forearm and lasted a month. Pain continued from the first over the outer part of the forearm towards the front; it was slight, but was increased on extension of the forearm. In addition, the patient noticed a numbness on the outer half of the flexor aspect of the forearm, extending, when the limb was cold, to the thumb and first two fingers. Flexion of the forearm was weak, but the hand and finger movements were normal. On being examined, the biceps was found a little wasted and obviously weak. Other than flexion at the elbow joint, no movement shewed any weakness, except, perhaps, supination. The biceps and brachialis anticus were flabby, in contrast with the coraco-brachialis and supinator longus. In the distribution of the cutaneous branch of the musculo-cutaneous nerve, there was anæsthesia to touch, hypæsthesia to heat and pain. The periosteal reflex of the radius was lost on that side. Reduction in response to galvanism and



faradism was found in the two affected muscles. An ascending infective neuritis was diagnosed.

Eleven similar cases of isolated paralysis of this nerve are on record; five of them were due to trauma, four to neuritis, as in the above case, and two were of uncertain origin. One of the cases of neuritis was due to gonorrhœa. The coraco-brachialis frequently escapes. It is to be observed, as Bernhardt pointed out, that slight paresis of this nerve may easily be overlooked, as the patient's work may not be interfered with.

ERNEST JONES.

**TOXIC POLYNEURITIS IN A PHTHISICAL PATIENT. (Toxische (271) Polyneuritis bei einem Phthisiker.)** WERTHEIM SALOMONSON, *Neurolog. Centralbl.*, May 16, 1906, p. 434.

A SCHOOL teacher, aged 21, was suddenly taken with paralysis of the lower limbs, and in a few days this was followed by weakness of both hands. The paralysis was preceded by pains in the calf and sole for two days. It came on a fortnight after ceasing a line of treatment for his pulmonary tuberculosis, consisting chiefly in the administration of phosphoric creasote. He had taken altogether 75 grains of this in seven weeks. The paralysis of the lower extremities was intense, and atrophy was present in the peronei and tibiales. Sensory changes were very inconspicuous. There were practically no skin changes. Sphincters unaffected. The etiology of the neuritis is discussed fully. After a perusal of the published cases, the author is doubtful of the very existence of a true tuberculous neuritis. A fortnight after seeing the above case he came across an exactly similar one from the practice of the same doctor, and heard privately that lately seven other cases of polyneuritis had developed while they were being treated by him with the same preparation. Although creasote and phosphorus are both innocuous drugs in fairly large doses, it is possible that, as happens elsewhere in chemistry, the combination of the two substances evolves a new body which is a poison to the nervous system. Lowenfeld published two years ago three exactly similar cases to the above and attributed them to the taking of phosphoric creasote. He suggests that the cord may be the part affected, especially as the motor symptoms are so much in excess of the sensory ones. Chaminet has also collected seven cases, and has no doubt as to the etiological factor. The cases recorded by Leyden in his classical work on polyneuritis as being due to phosphorus alone are quite different in their symptomatology from those referred to above. Babinski showed a case last year in which the cause seemed to be the manure which the man used at his work; he thought that some contamination of the manure with lead or

arsenic might explain the case, but Salomonson suggests that a derivative of the superphosphates which largely compose manure might with equal likelihood be the origin of the symptoms.

ERNEST JONES.

**TABES DORSALIS AND THE KNEE ANGLE PHENOMENON.**

(272) (*Tabes dorsalis und das Kniewinckelphänomen.*) J. G. ORSCHANSKY, of Charkow, *Neurolog. Centralbl.*, May 1, 1906, p. 401.

THE author recalls the fact that laxity of the knee ligaments is commonly to be found in tabetics. This may be thus demonstrated. In a healthy person who lies supinely it is impossible to lift a foot if the knee is fixed; in other words, as no hyperextension is possible, the leg and thigh form an angle of 180°. In many tabetics the foot can be raised under these circumstances, so that the leg may form at the knee an angle of even 16-20° with the bed beneath. This sign is never absent if the tabetic have ataxy, and corresponds with the more ataxic limb if it is only present on one side. It is found constantly in infantile paralysis and occasionally in myelitis, but never in cerebral paralyses. There is no correlation between the phenomenon and the appearance of lightning pains, or of other cutaneous sensory disturbances, but when unilateral it is found on the side on which the knee-jerk first fails.

Other changes about the knee accompany this phenomenon. If the thigh be fixed at the knee, the foot can be abducted, so that the leg and thigh form an obtuse angle. Further, the patella may be movable up, down, in or out, to an unusual degree, and it can be lifted from the lower end of the femur to such an extent that a finger may be insinuated between the two. The lower border of the patella is often thin and pointed. Observations similar to the knee phenomenon may be made at other joints, notably the ankle and elbow.

The phenomenon is doubtless due to the loss of muscle tonus and the diminution of elasticity of the ligamentous apparatus. It is possible that the joint changes are of high importance in the development of the tabetic ataxia.

ERNEST JONES.

**SOME SURGICAL COMPLICATIONS OF TABES DORSALIS.**

(273) ANTHONY A. BOWLBY, *Brit. Med. Journ.*, May 5, 1906, p. 1021.

THE moral of this clinical lecture is that the various complications are often transitory, and therefore deserve as careful surgical treatment in tabetic as in other patients. "Charcot's disease" of joints

may appear at any stage in tabes. Exceptionally, it is one of the earliest symptoms; sometimes it sets in twenty years after tabes is recognisable, and it may show itself at any intermediate period. The important point is, that the disintegration of the joint eventually ceases, and that, if the patient is kept at rest and the joint properly splinted while the destructive process is progressing, and if displacements are thereby prevented, he may be able to regain some use of the limb, and, indeed, may be able to dispense with splints, as the masses of new bone thrown out round the joint may form a natural splint.

"Spontaneous fracture" of bones from slight causes results from a similar dystrophy of bone. These fractures should be treated as in healthy people, for in many tabetic patients strong union occurs, and the tendency to fracture may pass away. Perforating ulcer of the foot may heal, and remain healed if kept clean and relieved from pressure; abstinence from alcohol is important in this condition. Other complications, such as gastric crises and incontinence of urine or fæces, may also be transient; and eye conditions, for example diplopia, squint, and grey atrophy of the disc, may cease to become worse.

Too bad a prognosis in tabes should not, therefore, be given; arrest of the disease may occur at any stage. Improvement of general nutrition is the main treatment; antisiphilitic remedies are useless. As many of the complications prove transitory, all should receive careful and appropriate surgical treatment.

W. J. STUART.

**MULTIPLE SCLEROSIS: A Contribution to its Clinical Course and (274) Pathological Anatomy.** E. W. TAYLOR (Boston), *Journ. of Nerv. and Ment. Dis.*, June 1906, p. 361.

THE view that multiple sclerosis is a very rare disease in America is not likely, in the author's opinion, to stand the test of further experience, although it is probable that it is less frequent than in Europe.

"Some of the apparent reasons for our neglect of the disease in America may be found in the predominance of out-patient clinics and the failure to follow cases to their end, with the consequent lack of autopsy reports. The insistence upon so-called cardinal symptoms, the neglect to recognise and properly interpret other obscure signs, the doubtful significance ordinarily attached to uncomplicated spasticity, and the personal bias in diagnosis, all account for the infrequency of diagnosis."

Dr Taylor refers to thirteen cases with autopsy reported in the American literature, and to these he adds eight which he has per-

sonally examined, three of which were studied in Germany. As evidence of the extraordinary interest which is being taken in disseminated sclerosis at the present time, the author has been able to discover eighty papers dealing with this disease in the literature of 1904 and 1905. In the cases which he has personally examined he has failed entirely to discover a symmetrical distribution of the sclerotic patches, except occasionally in what seemed to him to be a fortuitous way. He adheres to the opinion previously expressed that the grey and white matter are irrespectively involved. He was unable to discover that the grey matter formed the slightest barrier to the progress of the disease. Examination of several hundred specimens failed to establish any relationship between the vessels and lesions. The vessel walls often appear quite healthy, and where they show changes these are probably part of the general process.

A primary neuroglial overgrowth is held by many as the initial lesion. Borst holds that the process consists, firstly, in vessel changes; secondly, in a primary myeline degeneration; and thirdly, in a compensatory neuroglial overgrowth; and this writer admits the possibility of development of multiple sclerosis from a myelitis, as originally suggested by Leyden.

Bielschowsky regards the process as purely inflammatory, both neuroglia and nerve fibres being simultaneously involved.

E. Taylor's observations, however, are in perfect agreement with Müller's statement that no trace of an inflammatory reaction is to be seen.

Even though the process is not actually inflammatory with demonstrable lesions in and about the vessel walls, it is nevertheless both possible and probable in the author's opinion that the agent, whatever it may be, that produces the lesions, reaches the tissues through the blood or lymph channels. He remarks on the delimitation of the process in the individual patches, and suggests as an explanation the presence of an agent which spreads from a central focus until it exhausts itself. This view he thinks is far more reasonable than the hypothesis of a primary inflammation as that is usually understood.

Arguments are accumulating against a primary growth of neuroglia, and among these the frequent lack of evidence of extensive proliferation of the neuroglia, and the fact that the nerve roots often show degenerative changes without evidence of neuroglial proliferation may be cited. A more probable explanation would appear to be that a toxic agent of unknown character exists, which has a peculiar chemical affinity for myeline and possibly also for certain analogous material in the axone. A third alternative is a simultaneous action of the supposed toxic agent upon the myeline and the neuroglia, leading, on the one hand, to

a destruction of the myeline and coincidently acting as a stimulus to neuroglial proliferation. "The sequence of events is, however, impossible to determine with accuracy." EDWIN BRAMWELL

**THE CLINICAL SIGNIFICANCE OF THE CEREBRO-SPINAL (275) FLUID.** PURVES STEWART, *Edin. Med. Journ.*, May 1906, p. 429.

THE author describes the technique of lumbar puncture and the characteristics of normal cerebro-spinal fluid. He then considers the diagnostic data which may be obtained from the examination of the physical, chemical, bacteriological, and microscopical characters of the fluid, and finally refers to the therapeutic applications of lumbar puncture. In the case of one patient, comatose from cerebral thrombosis, he obtained normal clear fluid, whilst in another case with cerebral hæmorrhage the fluid was faintly straw-coloured.

Cryoscopy of the cerebro-spinal fluid has not been of great practical value.

Guillain and Parant have found in a series of sixteen cases of general paralysis that if the globulin of the cerebro-spinal fluid was precipitated by adding a saturated solution of magnesium sulphate and the clear filtrate boiled, a characteristic precipitate was obtained.

The demonstration of cholin in the cerebro-spinal fluid might occasionally be of value in diagnosis between hysteria and organic disease, but the method is somewhat too complicated and technical for the ordinary clinical observer.

In doubtful cases of tuberculous meningitis, inoculation of the cerebro-spinal fluid into guinea-pigs may prove of value.

After describing the method to be adopted in the cytological examination of the fluid, the author recounts his experience in 105 cases of various nervous diseases which he has recently examined. Whereas in the normal fluid not more than four cells should be seen, under a magnification of 400 diameters, in the deposit obtained after centrifuging 5 c.c. of the fluid for five minutes; in certain diseases this number, as is well known, is greatly exceeded. Thus there were more than 4 and less than 10 lymphocytes in the field in the following five cases: combined degeneration of pernicious anæmia (1 case), acute myelitis in syphilitic patients (2 cases), tuberculous tumour of cortex cerebri (1 case), persistent headaches of obscure origin (1 case).

In six cases there were from 10 to 25 lymphocytes to the field. Gumma of the spinal cord, Landry's paralysis, endothelioma of spinal cord, cerebral thrombosis with hemiplegia, post-epileptic visual hallucinations (?syphilitic), old syphilis + hemiplegia, functional type.

Moreover in the following conditions from 25 to 400 lymphocytosis, or even more, were present in each field: G.P.I. (12 cases), tabes (25 cases), gumma of crus cerebri, gumma of spinal cord, syphilitic hemiplegia (thrombosis), tuberculous meningitis (2 cases), glioma of corona radiata, posterior hydrocephalus relieved by operation.

In two cases of epidemic cerebro-spinal meningitis and meningitis from otitis media, a polymorpho-nuclear leucocytosis of 87.3 and 371 respectively was present. The lymphocytosis of tabes and general paralysis is uninfluenced by the most energetic anti-specific treatment. Syphilis, unless it has actively attacked the central nervous system, produces little or no lymphocyte increase in the cerebro-spinal fluid.

Regarding the therapeutic applications of lumbar puncture, the author points out that the lumbar puncture is often successful in alleviating the headache of intracranial pressure. Uræmic coma and convulsions are frequently relieved in a striking way. Reference is made to spinal anæsthesia.

Four special tables are given of lymphocyte counts in syphilitic patients without signs of nervous disease (12 cases), in tabes (25 cases), general paralysis (12 cases), and intracranial tumour (13 cases).

EDWIN BRAMWELL.

#### TRANSITORY HEMIPLEGIA, WITH NOTES OF TWO CASES.

(276) HAMILTON GRAHAM LANGWILL, *Scot. Med. and Surg. Journ.*, June 1906, p. 509.

THE author records in this interesting paper the history of two individuals who suffered from several attacks of hemiplegia of brief duration. "The patients were males, aged 57 and 71 respectively." In each patient there occurred a sudden seizure, having all the features of a typical hemiplegic attack without loss of consciousness, perfect recovery of motor power following in less than an hour, while in one case these attacks occurred on several occasions. In neither instance was there left any trace of motor enfeeblement.

The author lays special stress on the *suddenness* of the recovery and its *perfectly complete* character. After discussing the possible etiology of the condition, he comes to the conclusion that the paralysis was probably a consequence of a localised arterial spasm, and in this connection refers to two papers, one by Dr William Russell on "Cerebral Manifestations of Hypertonus in Sclerosed Arteries" (*Practitioner*, April 1906); the other by Dr R. A. Lundie, on "Transient Spasm of the Retinal Artery" (*Ophthalmic Review*, May 1906).

EDWIN BRAMWELL.

**INFANTILE CEREBRAL HEMIPLEGIA ; REFLEXES ABOLISHED**  
 (277) **IN THE LOWER EXTREMITIES AND LITTLE MARKED**  
**IN THE UPPER EXTREMITIES.** (Hémiplégie cérébrale in-  
 fantile ; réflexes abolis aux membres inférieurs, peu prononcés  
 aux membres supérieurs.) BOUCHAND (de Lille), *Arch. Gén. de*  
*Méd.*, May 15, 1906, p. 1236.

Writers on infantile cerebral hemiplegia describe spasmodic hemiplegia, hemiathetosis, and hemichorea, but scarcely mention forms where spasticity and abnormal movements are absent, and completely ignore the existence of cases in which the reflexes are abolished. The case here recorded is that of a boy aged ten years, the subject of left hemiplegia. He had been healthy until six years old, when he had an illness lasting six weeks, and apparently of the nature of a meningitis, from which he recovered completely. Nine months later, however, there was a sudden appearance of complete paralysis of the left arm and leg. Since that time considerable improvement had taken place. There had been no epileptic attacks. On examination he was noted to have deficient intelligence ; marked weakness and arrest of development of the left arm, and slight of the left leg ; but no affection of the face. The reflexes of the arms were not exaggerated ; those of the legs were absent ; there were no contractures. The electrical reactions of the muscles in both affected limbs were normal. The differential diagnosis is indicated, the literature of the subject reviewed, and probable explanations of the diverse manifestations of the disease discussed.

HENRY J. DUNBAR.

(278) **CEREBELLAR APOPLEXY.** STARR, *Med. Record*, May 12, 1906.

ALTHOUGH this condition is undoubtedly much rarer than cerebral apoplexy, the writer having found only four cases of cerebellar disease in 187 cases of apoplexy examined post-mortem, he urges in this paper the distinctiveness of its symptoms and the importance of its diagnosis.

Five cases of cerebellar apoplexy are detailed, the symptoms which they presented in common being vertigo of an extreme degree at the onset, and remaining to some extent as a permanent feature ; uncertainty of gait and staggering due to lack of balancing power ; an unnatural position of the head at rest ; nystagmus, vomiting, and headache.

The author attaches great importance to diagnosis, because he believes that cases treated as "stomach vertigo" in old people may be due to this condition, in which case the treatment for apoplexy is essential. He gives a copious bibliography.

JOHN D. COMRIE.

**INTRACRANIAL ANEURISMS.** BYROM BRAMWELL, *Clinical Studies*, (279) July 2, 1906, p. 289.

LARGE intracranial aneurisms are of rare occurrence. In this paper the author describes in detail and illustrates six such cases.

The following is a brief account of the leading features of the cases reported:—

*Case 1.* Large aneurism of the left internal carotid artery; severe headache, chiefly limited to the right side of the head; complete loss of sight in the right eye, loss of sight in the temporal part of the field of vision in the left eye; death from uræmia five years after the patient was in hospital and nine years after the first symptoms.

*Case 2.* Chronic dementia in a woman aged 43; syncopal attacks; headache; obesity; partial left-sided hemiplegia; difficulty of articulation and deglutition; progressive stupidity and drowsiness; death two years and nine months after the symptoms were first complained of; an aneurism, the size of a small orange springing from the right posterior cerebral artery, and projecting into the third and lateral ventricles; softening of the brain tissue around the tumour; hæmorrhagic pachymeningitis.

*Case 3.* Temporal hemianopsia; mental derangement; symptoms of a cerebral tumour; blindness; mania; gradual and progressive hebetude; death; an enormous aneurism of the right internal carotid within the skull; erosion of the sella turcica; pressure upon the optic nerves, optic chiasma, and optic tracts; softening of the brain tissue around the tumour.

*Case 4.* Aneurism of the basilar artery; meningeal hæmorrhage; immediate coma; death in fourteen hours.

*Case 5.* Aneurism of the left internal carotid artery; rupture; convulsion; violent delirium followed by pain in the back part of the head, stiffness of the neck, and retraction of the head. Second rupture a fortnight after the first attack; convulsions; delirium; coma; rapid rise of temperature; death.

*Case 6.* Aneurism of the right middle cerebral artery; rupture, attended with sudden pain in the head, and vomiting; gradual onset of coma and left hemiplegia four hours after the accident; rapid development of Cheyne-Stokes respiration; death from failure of the respiration six hours after the rupture.

In the same number the author reports three other cases of tumour in the same situation, viz. a large tumour of the pituitary body pressing upon the optic chiasma, a pituitary tumour without acromegaly, and a syphilitic gumma involving the optic chiasma, with recovery.

EDWIN BRAMWELL.



**EXTRA-CEREBELLAR TUMOURS.** BYROM BRAMWELL, *Clinical*  
(280) *Studies*, April 2, 1906, p. 254.

FOUR cases of tumour in the cerebello-pontine angle are described. One of these is of especial interest since the patient was operated upon and a growth of considerable size removed.

The following are some of the details of the case: The patient, a male, aged 27, when seen in November 1905, stated that for eighteen months he had been deaf in the left ear, and that soon after this was first noticed his eyesight became affected. Some time after this he found that his "gait" was unsteady. He had never had any tinnitus, vomiting, giddiness, or headache, although he occasionally felt a pain above the eyes. The family history was unimportant.

On examination it was noticed that he walked somewhat unsteadily, swaying especially to the left side. The head was turned slightly to the left, and the left ear depressed slightly towards the left shoulder. This position of the head might have been accounted for by a marked lateral curvature of old standing.

There was complete deafness both to aerial and bone conduction in the left ear. Well-marked optic neuritis was present, more advanced in the right eye. There was imperfect fixation on conjugate movement to the left, and slow, coarse nystagmus on attempting to keep the eyes in the position of extreme deviation to this side; lateral movement to the right was attended with quick nystagmoid jerkings.

Beyond these points nothing abnormal was detected. The opinion was expressed that there was an intracranial tumour probably situated in the left cerebello-pontine angle.

The patient was admitted to hospital on December 20th. At that time his gait was much more unsteady, and in addition there was a distinct upper neuron paresis of the right side of the face and some inco-ordination in the left hand.

The patient was subsequently operated on by Mr Cotterill in two stages. At the second operation on January 9th, 1906, after the dura had been opened it was found necessary to take away a large part of the left lateral lobe of the cerebellum in order to expose the tumour, which proved to be as large as a small hen's egg. The growth was removed, and on microscopical examination proved to be a fibroma. Unfortunately the patient died from respiratory failure eighteen hours after the operation.

EDWIN BRAMWELL.

**THE MENTAL SYMPTOMS OF CEREBRAL TUMOUR.** PHILLIP  
(281) COOMBS KNAPP, *Brain*, 1906, p. 35.

IN this paper the author has endeavoured to estimate the frequency of the occurrence of mental symptoms in cases of cerebral tumour from the clinical study of 104 cases, in which a growth of some nature was found at the autopsy.

Of these 104 cases, 40 were rejected on account of various complications, so that the author has based his investigations upon the study of 64 uncomplicated cases of tumour. In 58 of these cases (in over 90 per cent.), mental symptoms were noted; but the author holds that in every case of brain tumour presenting any cerebral symptoms, some mental symptoms can probably be discovered by a competent observer.

With regard to the nature of the mental symptoms occasioned by a growth within the skull, the author found that in 31 cases there was mental failure and dulness, the patients exhibiting varying degrees of languor, somnolence, apathy, mental torpor, failure of memory, and a general failure of all the mental functions, ending usually in complete stupor and coma.

Seven cases showed mental confusion and disorientation with mental failure, loss of memory, irrelevancy in speech, mild mental wandering, somnolence, and a dazed mental condition.

In 15 cases the mental impairment went on to actual states of delirium, and, even in some, to mania. Actual hallucinations and vague and unsystematised delusions were also observed.

Neurasthenic and hysterical states were observed in the earlier stages of the disease in some cases, but always developed more marked mental disturbance later on. True, *Wizelsucht* was not observed in any of the cases.

In about half of the cases the onset of the mental symptoms was early; confusion, mental dulness, somnolence, etc., characterised the earlier stages; delirium, violence, and profound stupor the later stages.

The cases examined did not bear out Schuster's figures with regard to the more frequent occurrence of tumours in the left side of the brain, but brought out the fact that in large growths the mental change was more marked.

The detailed examination of the actual symptoms in relation to the site of the growth did not give any support to the theory which seeks to establish one special psychical centre in the brain.

The investigations of the cases according to the period of development of the mental symptoms brought out the fact that in tumours of the corpus callosum or of the corpora quadrigemina the mental symptoms were of early development, and that next in importance, in point of time, come the temporal and frontal lobes,

followed by the other regions of the prosencephalon, the optico-striate region, the hypophysis, and the brain stem. The study of these cases lends support to the belief that the temporal lobe, apart from its association with the function of speech, has perhaps as important a share in the psychical functions of the brain as the frontal lobe.

Regarding the pathogenesis of the mental symptoms, various explanations have been brought forward. The mental disturbance may be due to (1) focal deficit or irritation, or (2) to increased intracranial pressure, or (3) to the formation of certain toxins in the brain, either from the new growth itself or by the disturbance of the circulation caused by the growth.

The author brings out the resemblance between the mental symptoms usually met with in cerebral tumour and those occurring in toxic psychoses, but was unable to find any special relation between the nature of the growth and the character of the mental disturbance except that in cases of sarcoma the delirious conditions were more apt to occur.

In conclusion, he points out that the cases he studied reveal nothing as to the nature or even the existence of toxins, but he considers that although the situation of the growth is often of influence in producing mental symptoms, especially in the early stages of the disease, and probably has an influence upon the nature of the symptoms, a combination of increased cranial pressure and the action of toxins is of greater importance, and in some cases may be the only factor to be considered in the production of such symptoms.

T. GRAINGER STEWART.

**A CONTRIBUTION TO THE STUDY OF CEREBELLAR TUMOURS  
(282) AND THEIR TREATMENT.** J. J. PUTNAM and G. A. WATER-  
MAN, *The Journ. of Nerv. and Ment. Dis.*, May 1906.

THIS paper contains reports of four cases of cerebellar tumour and three cases of tumour in the cerebello-pontile angle. Three of the cerebellar cases were successfully operated upon, and the remaining case died before an operation could be performed. Of the cerebello-pontile cases, one was operated upon and the growth removed, but the patient died suddenly four days later. One of the other two cases was wrongly diagnosed, and in the third case the presence of fits with a sensory aura in the arm led in the first instance to a diagnosis of tumour of the opposite Rolandic area. Both these cases show some of the points to be brought out in the differential diagnosis of such tumours, and further points of interest in this connection are illustrated by the reports of a case of tumour in the fourth ventricle and a case of pontine tumour. The authors

strongly advocate operative treatment, and condemn the practice of treating such cases with iodide for long periods; such treatment never being successful, and often rendering the chances of successful operation less hopeful. The operations were always carried out in two stages. The chief danger attending operation is failure of the respiratory centre. This occurred in two cases: in both artificial respiration was resorted to, and the patients made a complete recovery.

T. GRAINGER STEWART.

**THE SIGNIFICANCE OF JACKSONIAN EPILEPSY IN FOCAL  
(283) DIAGNOSIS, WITH SOME DISCUSSION ON THE SITE  
AND NATURE OF THE LESIONS AND DISORDERS  
CAUSING THIS FORM OF SPASM. MILLS, *Boston Med.  
and Surg. Journ.*, April 26, 1906.**

THE writer defines Jacksonian epilepsy broadly as "monospasm or hemispasm due to cortical or cortico-subcortical discharge," but states that it is by no means true that it is always, or even nearly always, due to gross lesion of the motor zone.

He records, in the first place, several cases of successful operation for tumour, cyst, or gumma of the cortex, in which the focal diagnosis had been correctly made by observation of the parts involved in the Jacksonian attack, but the greater part of this lengthy paper is taken up by an examination of the fallacies which may interfere with diagnosis. He considers that this symptom may arise in the following five sets of conditions: (1) Tumours in other parts of the brain than the motor cortex. The writer records two cases of this condition produced by tumour in the cerebello-pontine angle, and quotes two cases of cerebellar tumour recorded by Collier, in which the symptom is attributed to accompanying hydrocephalus. On the other hand, the writer states that he has had several cases of parietal tumour involving the motor area in which Jacksonian epilepsy was not present, and he believes that destruction of the sensory cortex and subcortex before involvement of the motor fibres or cortex gives a certain immunity from spasm. (2) The symptom may also be due to other lesions of the motor cortex besides tumours, *e.g.* depressed fractures, localised meningitis, meningeal or cortical hæmorrhage, focal encephalitis, or focal necrosis from embolism or thrombosis. He discusses the diagnosis of these and quotes numerous cases. (3) Jacksonian epilepsy may occur in toxic and other diseases with no demonstrable focal lesion of the brain. The writer states that one of the most striking cases of this symptom that he has seen occurred in a case of diabetes, while it is also occasionally a sign of uræmia or of acute alcoholism. (4) Peripheral irritation

almost anywhere in the body may cause a convulsive attack, and this in rarer instances may assume the Jacksonian type. In this connection the writer records a case due to a fibroma in the palm of the hand. (5) Jacksonian epilepsy may be simply a part or the entire expression of a case of idiopathic epilepsy.

Numerous references to the literature of the subject are also given.

JOHN D. COMBIE.

**THE SOMATIC EVIDENCES OF SYPHILIS IN PARETICS.**  
(284) WINFIELD (of Brooklyn), *New York State Journ. of Med.*, May 1906.

THERE were 241 cases of general paralysis examined from six of the State hospitals in the neighbourhood of New York. All but six were males. The reason why more females were not examined was because the cutaneous evidences of syphilis in women are not, as a rule, as well marked as in men; consequently it was thought best not to submit the females to the rigid examination necessary. All were examined for external evidences of syphilis, irrespective of any history of the disease: 165 had scars and markings that were typical of cutaneous syphilis; 76 showed no external evidences whatever. The hospital histories showed, however, that 28 of the 76 negatives gave an undoubted history of syphilis. If these 28 are added to the 165 exhibiting evidences of cutaneous syphilis, there would be 193 out of 241, or about 80 per cent., that had had syphilis.

Cutaneous phenomena were more common and more pronounced among the patients confined in the rural hospitals than among those confined in the metropolitan hospitals. This is explained by the ease of obtaining treatment at the free hospitals and dispensaries of a metropolis.

Of those of foreign birth, the greatest number were Germans, 31; Irish, 17; Russian, 8; Italian, 5; English, 4; Austrian, Hungarian, and Swedish, 2; French, Roumanian, Finnish, each 1.

Concerning the distribution of scars, Winfield states "46 had scars on the penis; 77 had scars on the legs and thighs; 73 showed markings over the trunk; 18 over the arms and hands; 14 had inguinal scars (bubonic); 30 had general adinitis; 3 had mucous patches on the scrotum; 7 had scars and topi on the scalp; 4 showed scars and destruction of the lips, nose, and palate; 20 had nodes along the tibia; 5 showed ptosis, 4 blindness from optic neuritis, and 2 showed the stigmata of congenital syphilis.

C. H. HOLMES.

**THE SUPPOSED IMMUNITY OF SYPHILITIC ARABS REGARD-**  
**(285) ING GENERAL PARALYSIS.** (*La légende de l'immunité*  
*des Arabes syphilitiques relativement à la paralysie générale.*)  
MARIE, *Rev. de Méd.*, May 10, 1906.

THIS is a contribution to the question of the relation subsisting between syphilis and general paralysis, as well as a refutation of the commonly accepted opinion that general paralysis is not frequent among the Arabs. The inquiry is based upon the statistics of the admissions to the Abbassieh asylum for insane at Cairo, numbering 3600 in ten years. The writer found here that the general paralytics made up 6 per cent. of the admissions, and though this is but half the rate for Paris, it is greater than that found in provincial French asylums. Taking two years from the middle of the decade, 1900 and 1901, the numbers of Arabs were respectively 21 and 23 out of totals of 25 and 35 general paralytics admitted to the asylum. As to syphilis, while its prevalence in the cases throughout the asylum was only 12 per cent., among the general paralytics 79 per cent., or about six times as many, were proved to be syphilitic.

Some interesting facts are also given as to the native treatment of the insane among the Arabs, and reasons advanced why in Tunis and Algeria general paralysis appears to be less prevalent than in Egypt.

JOHN D. COMRIE.

**COMMUNICATION ON THE "HYSTERIA" OF ANIMALS.** (Mit-  
'(286) teilungen über die "Hysterie" der Tiere.) J. MAINGER,  
*Neurolog. Centralbl.*, May 16, 1906, p. 438.

THE literature on this subject is very sparse. This is doubtless in part due to the attitude taken by most veterinary surgeons towards the study of the mind in animals. Yet if only from a theoretic standpoint the cases of hysteria are of great interest, occurring as they do in minds which are relatively simple. After a critical review of the literature, Mainger affirms that only five true cases have been published; most of those labelled hysteria in animals, even the oft quoted one of Charcot's, are really cases of other psychoses. Mainger has personally observed three other cases which he now publishes.

Case 1. Female fox-terrier, aged 2. It suffered first from a broken pelvis through a horse kick, and limped in the right hind leg for six months. Then the right fore leg was broken, and later the dog was twice run over by a bicycle with no ill result. One day when scrambling up a loose pebbly slope, which was too steep to allow progress, it slipped back in spite of all its efforts, and

when reached by its master had first the right, then both hind legs strongly contracted. This was followed by opisthotonus with no loss of consciousness. It was unable to stand or move the hind legs for five minutes, and then unexpectedly recovered power. It died of poisoning a year later and no changes were found in the nervous system.

*Case 2.* Male Bolognese dog, aged 1. Convulsive attacks occurred not infrequently. These were epileptiform in nature and were followed by a temporary amnesia, so that the animal would snap at its master. They were brought on chiefly by a fear, akin to agoraphobia, that occurred on the dog being forced to enter an open space, such as a field. Occasionally exciting episodes, such as losing its master, being in a crowd of other dogs, etc., would also induce attacks.

*Case 3.* Fox-terrier, 18 months old. Attacks occurred until the age of 2. They consisted in extensor spasm with opisthotonus; staring eyes; consciousness was retained. They were induced by anxious moments, such as losing his master or finding himself the centre of interest of strange dogs. The master, an electrical engineer, was advised to use faradism, but this made the dog worse, invariably inducing an attack.

The psychical origin of the attacks in every case is insisted on by the author, who then discusses in an interesting way the bearing of the cases on human hysteria and especially on the treatment of this.

ERNEST JONES.

**ISCHEMIC MUSCULAR ATROPHY, CONTRACTURES, AND  
(287) PARALYSIS.** ALEX. HUGH FERGUSON, *Ann. of Surg.*, April 1906, p. 599.

THE changes described are those in muscles, following more or less complete arrest of their blood supply. A brief résumé of the causes, symptoms, pathological anatomy, and course of the condition is given. Tight splinting was the cause in the two cases referred to in this article. The sequence of symptoms is briefly as follows—paræsthesia, pain, cramps, loss of electric irritability, muscular rigidity, and contractures followed by flaccidity, and ultimately permanent contractures from muscular atrophy and overgrowth of fibrous tissue. In both cases operated upon by the author there was marked flexion of wrist and fingers, which could not be overcome by any ordinary force; the muscular bellies, especially of the flexors, were atrophied; and there was more or less paralysis, with sensory and vaso-motor disturbances. The operative procedure consisted in free exposure of the contracted flexors, the bellies and tendons of which were found to

be matted together and extremely atrophied, followed by separation of the adhesions, and lengthening of each of the musculo-tendinous structures by one of the ordinary surgical methods. The median and ulnar nerves were freed and stretched. Finally, sterile olive oil was poured over the tendons to prevent immediate re-adhesion and the wound was closed. The post-operative reports show that complete passive movement and a fair amount of voluntary movement became possible, the result being a great improvement, though not by any means a restoration to a normal condition. The nerves were so shortened and fixed that forcible extension of the limb would probably have ruptured them. The author thinks that resection of part of the bones, with consequent shortening of the forearm, might be a better operation than tendon splicing. Various references to the literature of the condition are given.

W. J. STUART.

**ON UNILATERAL LOSS OF, AND ON SUBSEQUENT RETURN**  
(288) **OF A LOST KNEE-JERK.** (Ueber einseitiges Fehlen und über die Wiederkehr des verschwunden gewesenen kniephänomens.)  
GASTON WEHRUNG, of Bonn, *Neurolog. Centralbl.*, May 1, 1906, p. 391.

THIS contribution is based on three cases observed with care in Westphal's clinic, and subsequently examined post-mortem.

*Case 1.* Woman, aged 40. Complaint chiefly was of psychical and intellectual symptoms. Pupils were unequal and quite fixed. The left knee-jerk was very active, the right absent, even with Jendrassik. This condition remained absolutely the same during the ten months she was under observation. The left limb was spastic and the right flaccid. Post-mortem, there was found in the thoracic and upper lumbar region a slight degeneration at the side of the posterior columns, especially in Westphal's root entry zone, on the right side, whilst on the left the corresponding part was healthy. There was also some degeneration of the pyramidal columns, most marked on the left side. The clinical signs were thus explained.

*Case 2.* Woman, aged 31. Case similarly of paralytic dementia, with unequal and fixed pupils. Rombergism. No Babinski. Knee-jerk absent on both sides. After a second attack of right-sided paresis, the right knee-jerk returned. The left was also very slightly observable for a short while. The right knee-jerk was present till death, four months later. Post-mortem: (a) Van Gieson. In cervical cord, slight fibrosis in both pyramidal columns. Many small hæmorrhages in right posterior horn. In thoracic cord, best seen about the middle, is a symmetrical area of degeneration, marking out the comma tract. Otherwise no fibrosis worth mentioning.



in the pyramidal tracts of the thoracic or lumbar cord. Slight change, equal on both sides, in posterior columns of lumbar cord. Four or five hæmorrhages in posterior horn and Clarke's column at the level of the first lumbar segment on the left side. (b) Pal-Weigert. Similar to above. (c) Marchi. Recent degeneration in posterior columns only.

*Case 3.* Man, aged 38. Also a case of paralytic dementia. Pupils unequal and fixed. No Rombergism. Knee-jerks and Achilles-jerks absent. After an attack, which was not followed by paresis, the right knee-jerk reappeared, and ten days later the left also. In a fortnight they had again disappeared, but after another attack, a week later, the right returned and remained active for two months, failing only two days before death. Post-mortem, no changes were found in the pyramidal tracts by any method, save Marchi. Moderate degeneration of the posterior columns was found throughout the cord; this, in the lumbar region, was more marked on the left side, especially in the root entry zone. By Pal-Weigert staining, the fibres entering Clarke's column were seen to be degenerated on the left side, not on the right.

From a consideration of these cases, and others that the author reviews from the literature, the conclusion is reached that retention of the knee-jerks in tabes depends on the escape of the root entry zones from degeneration, and that their reappearance can occur only when these zones are not severely affected. Pick's hypotheses on the subject are discussed, and the one favoured that assumes disappearance of the knee-jerk to depend on obstructions in the nerve path, and reappearance on the removal of these obstructions.

ERNEST JONES.

#### DEAFNESS DUE TO HYSTERIA AND ALLIED CONDITIONS.

(289) P. M'BRIDE, *Edin. Med. Journ.*, May 1906, p. 391.

THE author discusses the subject of deafness due to hysteria and allied conditions, and points out the difficulty of being sure of the diagnosis in such cases until the hearing is restored.

In one of the seven cases recorded (No. 3) it was noted that a tuning fork placed on the vertex was heard less clearly when the ears were closed. Case 4 had already suffered from hysteria when first seen, and the subsequent notes showed that the hearing power returned and went away again on several occasions. Case 7 is interesting, because it showed a diminution of sensibility of the auditory canals and tympanic membranes, and also because the patient stated at the beginning of the examination that the tuning fork, when placed on the vertex, was heard best by the left ear,

whilst at the end of the examination she located the sound in the right ear.

Dr M'Bride next discusses the literature of the subject, and quotes Politzer as saying that hysterical deafness is usually confined to one side, that bone conduction is lost, and that this kind of deafness is associated with diminished sensibility.

Dench is also referred to, especially with regard to the perception of high and low notes—"Upon one side the upper tone limit will be found greatly reduced, while the organ of the opposite side will perceive the highest tones of the scale with ease. On repeating the experiment the condition will be exactly reversed, and this alternation may be repeated several times during the examination."

According to Oppenheim, as quoted—"Unilateral hysterical deafness does not interfere with hearing, as a whole, to any great extent . . . often they are not aware of the deafness . . . bilateral deafness is usually only a transient phenomenon."

Boulay and Marc'hadour have described cases similar to those given by Dr M'Bride in his paper: he gives the following extract, among others, from their writings:—"But whether the dynamic trouble occupies the whole scene, or whether it is superadded to an affection of the ear, it is always possible to track it, for it has from its beginning a special stamp . . . diminution or absence of sensibility of the meatus and membrane, disappearance of perception for high and medium tones, absence of subjective trouble, diminution or disappearance of bone conduction . . . in hysteria the central neuron has lost its connection with the peripheral . . . a corollary—the absence of subjective phenomena."

The author states that he cannot attempt to give a résumé of Gradenigo's work, though he considers it the most complete on the subject: some of the more important points are, however, clearly noted.

Finally, in investigating one of these cases, Dr M'Bride suggests the following points as worthy of attention:—

1. Is there any marked discrepancy between the history and the results afforded by objective examination?
2. Do the history and manner of the patient suggest hysteria?
3. Do history and results of examination accord with any of the recognised forms of organic disease?
4. Is there any evidence of sudden improvement of hearing when patient is interested?
5. Do repeated hearing tests give the same result?
6. Is there any anomaly of hearing?

He points out, in conclusion, that there is no question of prognosis in these cases, for the diagnosis itself is only made certain when there is no longer a question of prognosis—in other words, when hearing has returned.

J. S. FRASER.

**DEVELOPMENTAL ALEXIA (CONGENITAL WORD-BLINDNESS).**

(290) By EDWARD JACKSON, M.D., *Am. Journ. of the Med. Sc.*, May 1906, p. 843.

THIS condition was first described by Hinshelwood in 1900. Other cases have been reported, the number including the author's cases, now amounting to nineteen.

The present article describes two cases.

*Case A.* A girl, æt. 11 years, came in April 1900, for headache, eye-strain, and special difficulty in reading. General intelligence good. Hesitates and miscalls the larger letters on the test-card, as much as the smaller ones. Under special tuition at home the difficulty gradually decreased. February 1900.—She now reads letters without hesitation. Reads a good deal and enjoys it. January 1905.—Her ability to read seems quite normal.

*Case B.* A boy, æt. 7 years, brought because his teacher says "he cannot see letters either near or far." After a year at school he only recognises with certainty O and H. Has full vision with each eye. Slight hypermetropia. Seems bright and intelligent. Recommended special tuition at home, and on February 18th he is said to be making good progress with his reading, and to have no difficulty with figures.

The writer gives references to, and a brief summary of, the seventeen previously described cases. He does not seem to be aware of the cases described by Dr Kerr in his last Report as Medical Officer to the London School Board, nor those described by Dr Sydney Stephenson in the "Reports of the Society for the Study of Disease in Children," Vol. iv. W. B. DRUMMOND.

**PSYCHIATRY**

**THE COMING OF PSYCHASTHENIA.** G. A. BLUMER (Providence, (291) R.I.), *Journ. of Nerv. and Ment. Dis.*, May 1906.

PSYCHASTHENIA is a condition, not a disease, and its interpretation should be widely extended. It is an exhaustion, and not a defect psychosis, although it may be evolved from an inborn weakness and instability. Symptoms common to this condition are not infrequent in the early stages of paresis, dementia præcox, manic depressive insanity, and senile dementia.

Janet makes five divisions based upon the degree of the morbid mental condition: (1) the simple neurasthenia with physical and moral depression, but without any accompanying sense of disease; (2) the patient who feels acutely and suffers from his state of depression, but shows a tendency to exaggerate and to generalise;

(3) one who has crises of agitation and anguish; (4) one who exhibits tics, phobias, or mental manias; (5) one who summarises all preceding disorders in obsessional ideas of shame, crime, sacrilege, expressed by crisis or continuously.

Janet makes three divisions according to symptoms into (1) the motor, (2) the effective, (3) the intellectual fields. Blumer, in conclusion, refers to the stirring events in psychiatry during the past ten years; the introduction of the term dementia præcox, the readiness with which it has been accepted, and its comprehensiveness. He urges that the Americans do not expose themselves to the criticism of a "cynical confrère" over the seas, who says that in America "everything is dementia præcox from idiocy to general paralysis."

C. H. HOLMES.

**THE FEELING OF UNREALITY.** F. H. PACKARD (of Boston), (292) *Journ. of Abnor. Psychol.*, June 1906, p. 69.

A BRIEF statement of the views held by previous writers upon the loss of feeling of reality, introduces the case of an unmarried woman, æt. 34, in a third attack of depression of sudden onset, who, at admission, sixteen months after beginning of attack, complained of a feeling of inadequacy, and especially of the feeling of unreality related to external objects, her body, and personal identity; this being associated with a belief in, and memory of, reality. Examination proved no disorder of the primary sensations. Tests with associations of simple words (nouns with appropriate adjectives and nouns associated by contrast) led to no definite results. But upon being given a simple story to read she found great difficulty in analysing and grasping the point; each component simple sentence had to be gone over and "visualised." Upon this basis an apperceptive disorder is considered as having been demonstrated for this group of cases. The lack of prompt apperception leads to a feeling of lack of familiarity or to the feeling of unreality. This same line of explanation is brought to apply to the association and apperception of matters in the somato and autopsychic spheres of mental activity as well as in the aldopsychic.

G. Y. RUSK.

**NATURE OF DEMENTIA PRÆCOX.** WALKER (Dixmont, Pa.), (293) *New York Med. Journ.*, May 19, 1906.

IN a mental disorder so clearly founded upon original inadequacy of constitution of the involved organ, one expects to find anomalies of structure indicative of this defect. Walker finds these abundantly present in his own cases; imperfectly formed and asymme-

trical ears, palatal deformities, and facial asymmetries have been present in all of his cases.

The mental symptoms are associated, as a rule, with bodily states which are both significant and explanatory of the nature of this disorder—poor appetite, loss of weight, anemia, headaches, insomnia, and alteration and perversions of sensibility are among the bodily symptoms mentioned.

The dementia is the one constant characteristic which unifies all forms of this disorder, and this implies structural changes (Cowles). Structural defect is first manifested by functional failure, and to be able to interpret the early symptoms of functional failure is of extreme importance in order that errors in nutritive functions, etc., may be corrected. The histories of several cases are briefly reviewed, so as to emphasise the important facts in the etiology, and the recognition of the earliest symptoms of the disease. Whether manifested at puberty, during adolescence, or in later adult life, and whether traceable to toxic agents or influence of stress and exhaustion, dementia præcox must be viewed as a representative of a relatively late stage of mental disorder, originating in an inherent defect in the vitality of the higher brain cells. The apparent recovery in a small percentage of cases is due to the relatively greater degree of vital endurance of the brain cells, or to a less marked involvement of the lower vegetative and organic functions. Under the favourable circumstances of early diagnosis and treatment, these cases are brought back to a normal state, because of the greater recuperative energy and reparative powers.

When once the disease is established it is progressive, and it leads ultimately to deterioration, which varies from a slight stunting of mental development to a reduction as extreme as that which follows any of the degenerative psychoses. There is no means by which one may accurately foretell the outcome of a given case.

C. H. HOLMES.

#### **DIAGNOSTIC ASSOCIATION STUDIES, SIXTH CONTRIBUTION.**

##### **(294) PSYCHOANALYSIS AND ASSOCIATION EXPERIMENT.**

(Diagnostische Assoziationsstudien. VI. Beitrag. Psychoanalyse und Assoziationsexperiment). C. G. JUNG (of Zürich), *Journ. f. Psych. u. Neur.*, March 1906.

THE author gives the detailed examination of a patient suffering from a psychogenic neurosis by means of his association method, and of Freud's psycho-analytic method. The contribution is especially useful as the analysis of the individual associations is fully given, and the value of the method employed clearly demonstrated.

The patient was a woman of 37, a teacher, who came to be treated by hypnotism for sleeplessness; in addition, she complained of inner unrest, impatience, and irritability. At the first interview Jung was struck by her peculiar manner; she spoke past him instead of addressing him directly; she made frequent restless, twitching movements, one of the most characteristic of which was the abrupt advancing of the lower part of the abdomen; she said that she felt that she was incurable, would go insane. Only after strong urging did she confess with much blushing and evidence of disinclination, as if telling some sexual experience, that she was unable to go to sleep because of the besetting idea, which would continually crop up, that she could not sleep, and would not until she was dead. She was also brought to confess that she had other obsessions. She denied, and apparently frankly, any sexual incident in the past. Owing to her condition, an attempt at hypnotism was not successful; accordingly, Jung applied his association method in order to get some clue to the disorder. He records the 100 association words used and the reactions of patient, and shows how these reactions pointed unmistakably to the existence of an underlying sexual complex. To those unfamiliar with the method of Jung and his results, the conclusions may seem unsound and almost fantastic, but his conclusions are the result of a very thorough study of associations, various parts of which have been published in this journal. The average reaction time of patient was 2.4s., while a person of her education would normally have a figure near 1.5. Examining those reactions which showed marked retardation, more than one complex was elicited, the most prominent being the sexual complex. The physician told patient that her obsessions were merely the distorted expression of an underlying sexual current, and that she was beset by sexual ideas. Patient denied this earnestly, and with considerable emotion. In the next interview Jung applied Freud's psycho-analytic method, or the method of spontaneous association; this method consists in merely inducing the patient to tell without reserve whatever chances to crop up in her mind. For the first half hour patient gave practically little, but before the sitting was concluded she had told of an indecent proposal made to her several years previously; the disclosure was made unwillingly, after much internal struggle, and was accompanied by the same gestures as had struck the physician on the first interview. During three weeks, on alternate days, the interviews were continued, and by the end of that time patient had disclosed a fund of sexual memories dating back to her childhood. In the store of images thus disclosed, the act of coitus was the most prominent, and was the centre round which later associations had disposed themselves. Patient herself had not been conscious of this master

motive at work in her sub-conscious life. The governess had swept the chambers of her mind clean, but did not know the nature of the contents of the cellars, nor of the influence of the latter upon her conscious life. The existence of a psychic trauma long before puberty in her case confirms Freud's view of the great importance of this as a dissociating factor in one's mental life.

For some time after these interviews patient was tormented with obsessions of a sexual character, but later all these obsessions and the secondary obsessions, which had developed without her being conscious of their relation to her sexual life, disappeared. Her sleep was restored, and only occasionally was disturbed through sexual ideas. Jung sums up as follows: (1) in psychogenic neuroses the complex which is disclosed by the associations is the cause of the disease (the predisposition being supposed); (2) the association method enables one to discover the pathogenic complex, and to shorten Freud's psycho-analytic method; (3) the association method shows us that hysterical phenomena and obsessions may be derived from the one complex; bodily and mental symptoms are merely symbolical derivatives of the pathogenic complex.

C. MACFIE CAMPBELL.

**FRAGMENT OF AN HYSTERIA ANALYSIS** (*Bruchstück einer (295) Hysterie-Analyse*). S. FREUD (of Vienna), *Monatschr. f. Psych. u. Neur.*, Oct.-Nov. 1905.

IN this communication, with its modest title, Freud does more than merely analyse the mechanism of the disorder in the case which he describes; he continually diverges to wider issues, and with the one case as his text, he gives his general views on the psychopathology of hysterical and allied disorders. He again emphasises his belief that a psychic trauma of a sexual nature before puberty is at the root of the hysterical dissociation; he traces out through all their subtle ramifications the submerged mental complexes and their manifestations, both physical and psychical, in the conscious life of the individual; he lays stress on the existence at an early age in the psychical constitution of the individual of homosexual tendencies, which may not be suspected by the owner, but which no less modify his reactions. Gestures and actions of apparently the most trivial import are explained in their symbolic meaning with reference to subconscious trends and groups of associations; and dreams are subject to rigid analysis, and shown to be equally determined, even in detail, by the same subtle influences.

Freud's interpretations are subtle, far-reaching, and so bold that they make the reader gasp; but his strenuous endeavour to push

his analysis as far as possible, and to demonstrate universal laws in what appear the most capricious and evasive aspects of mental life, is stimulating from the point of view of psychological insight, and has already given valuable clinical results.

Such work necessitates a most searching investigation into the sexual elements in our mental constitution, and it is unfortunate that the author has to spend time in defending this aspect of his work against those who fail to take a sufficiently serious grasp of the problems involved.

C. MACFIE CAMPBELL.

**IDIOCY AND A CEREBELLAR LESION. AMELIORATION OF**  
(296) **SYMPTOMS.** (*Idiotie et lésion cérébelleuse. Amélioration des symptômes.*) JULES VOISIN, ROGER VOISIN, et A. RANDU,  
*Arch. Gén. de Méd.*, May 29, 1906, p. 1365.

WHEN idiocy is associated with cerebellar symptoms it is extremely rare for any improvement to take place, and the case here recorded is therefore of some interest. It is that of a girl who was not thought to be in any way abnormal until defects of speech and walking drew attention to her condition. The birth had been natural, but at the fourth month of intra-uterine life the mother had received a severe emotional shock resulting in syncope. On admission to the Salpêtrière at the age of  $7\frac{1}{2}$  years the patient could only speak one or two words, did not understand what was said to her, could neither walk nor stand erect, and was generally helpless. The knee-jerks were diminished. Since then—twelve years ago—she has steadily improved both physically and mentally. The intelligence, however, is still poor, and her gait is now markedly cerebellar; speech slow and syllabic, knee-jerks exaggerated, and nystagmus present. The writers discuss the differential diagnosis from hereditary cerebellar ataxia, Friedreich's ataxia, and disseminated sclerosis. They consider the condition one of combined lesion of the cerebrum and cerebellum, dating probably from foetal life, the exact nature of which it is impossible to determine.

HENRY J. DUNBAR.

**UEBER DIE STIMMUNGSSCHWANKUNGEN DER EPILEPTIKER.**  
(297) Prof. Dr GUSTAV ASCHAFFENBURG, *Sammlung zwangloser Abhandlungen aus dem Gebiete der Nerven- und Geisteskrankheiten*,  
Bd. vii., H. 1. Carl Marhold, Halle a. S., 1906, pp. 55, price M. 1.60.

IN this important and extremely interesting paper, Prof. Aschaffenburg seeks to show that fluctuations in affective tone form a specific symptom of epilepsy, a symptom which on account of



the frequency of its appearance is of the greatest diagnostic value, and, further, that neither convulsive or vertiginous attacks nor emotional depression are the cardinal symptoms of epilepsy, but periodic fluctuations of psychic equilibrium which, according to the participation of the central nervous system, may lead to lighter or severer disorders of consciousness, and which may, *but also may not*, be accompanied by convulsions. This widened conception of epilepsy is, of course, of the utmost importance from its bearing on conduct, and particularly criminal conduct. It will be remembered that in 1895 Aschaffenburg read a paper, published in the *Archiv. für Psychiatrie* (Bd. xx., S. 955), on "Certain Forms of Epilepsy," in which he described these periodic fluctuations and, following Hoffmann, who first (in 1862) used the term "epileptic equivalent," strongly maintained their epileptic nature. His observations were mainly made upon dipsomaniacs under detention and the patients were subject to periodic attacks of bad spirits, during which they became tired, depressed, meditated suicide, had impulses to wander and escape, were fearful and apprehensive without cause, and suffered from headache, but without either delirium or obvious intellectual disturbance. Outside the Institution the patients sought to relieve their depression by recourse to alcohol, which was quickly followed by stuporous or dazed states (*Dämmerzustände*), indistinguishable from those of epilepsy proper. By a comparison of these results with those obtained at the Heidelberg Klinik for insane epileptics requiring compulsory detention, an admittedly circumscribed class, the author's opinion that these fluctuations were of epileptic nature, and may either accompany or replace the classical type of attack, was corroborated. Prof. Aschaffenburg's views have been widely criticised, and on account of the partial nature of his former material he now, on the basis of extended studies and with material to which exception cannot be taken, again opens up the whole question. The two questions which he sets himself to answer are:—

- (1) Are these fluctuations characteristic symptoms of epilepsy?  
and
- (2) In what way are they distinguished from similar disturbances in the psychopathies in general?

Prof. Aschaffenburg finds the answer to the first question comparatively easy, and he shows by means of tables, firstly, with regard to epileptics with convulsive attacks; and secondly, with genuine epileptics having no convulsions during their observation, that with the exception of vertiginous attacks, the periodic fluctuations form the most characteristic feature, the actual order of frequency in 50 cases investigated, entirely unselected, being vertigo (74 per cent.), periodic attacks of depression

or excitability (70 per cent.), petit mal (58 per cent.), "fainting attacks" (44 per cent.), convulsive seizures (42 per cent.), stuporous states (36 per cent.), wetting beds (28 per cent.), and impulses to escape or wander (18 per cent.). The striking feature of these results to which Prof. Aschaffenburg directs attention is the place in the list which convulsions take. All doubtful, or only slightly marked, fluctuations were excluded from the records, and, as the patients were nearly all prisoners undergoing sentences for moral and other delinquencies, and thus under constant and minute supervision, any convulsive seizure, however transitory, would be bound to come under notice. Thus 70 per cent. of the total epileptics showed, to quote Prof. Aschaffenburg, marked variation of emotional tone as epileptic equivalents. These fluctuations were almost constantly of sudden onset, characterised subjectively by fear, feelings of impending disaster, of "internal irritability," of nostalgia and a desire to wander, of "having done evil," of the desire, in some cases actually attempted, to commit suicide, of great difficulty in thinking—never, however, proceeding to marked disorientation; and, objectively, of delay in reaction-time to questions, memorial lacunæ, and increase of prison offences, insubordinate conduct, etc., with, as bodily symptoms during these periods, pulse acceleration, palpitation, profuse sweating, diarrhoea, hemicrania, changes in body-weight, hand-tremor, dilatation and comparative immobility of pupils, and changes in the individual's character, passing off entirely till the next attack. A point which is barely touched upon, and which should have been thoroughly investigated, is that of the patients' subsequent degree of memory as to the attacks and the occurrences during these periods. Turning to the second question, that of the differentiation of the fluctuations from similar states occurring in the course of other psychopathies, Prof. Aschaffenburg finds this much more difficult of solution, particularly as between epilepsy and congenital weak-mindedness, as the conditions are so frequently combined. In this relation he recalls an instructive case of an imbecile, in whom the author had diagnosed epilepsy entirely from the periodic fluctuations, and in whom later classical epilepsy developed. The whole question is one of great interest, and, from the criminological point of view, of much importance, and it seems probable that many of the periodic outbursts of temper, vagaries of conduct, and insubordination characteristic of weak-minded prisoners, are more easily explicable on Prof. Aschaffenburg's hypothesis than on any other.

R. CUNYNGHAM BROWN.

## Reviews

**UEBER ROBERT SCHUMANN'S KRANKHEIT.** P. J. MÖBIUS.  
Carl Marhold, Halle, 1906, M. 1.50.

THIS little book is an example of the attempts, frequent at the present day, to establish a retrospective diagnosis from the study of documentary evidence. The writer has carefully analysed all the Schumann literature, including Litzmann's recent book on Clara Schumann, and the letters and diaries it contains. From these he finds that both Schumann's parents were "nervous," that his only sister died from a severe form of dementia præcox, and that all his brothers died during early manhood. As a boy, Schumann was ambitious, generous, and good-tempered. In his youth he was distinguished by his artistic talents, and by a peculiarly feminine cast of temperament. His parents were quite unmusical, so that Schumann's musical genius is somewhat of an enigma. The writer regards it as a sign of abnormal mental disposition—as the pearl in the shell, for the production of which the whole must suffer.

During the first twenty years of his life, Schumann had no serious illness, but after that period, without any apparent cause, there were occasional attacks, and in his twenty-third year the disease assumed a graver form. The main symptom was a condition of unreasoning anxiety. During one night of frightful mental anguish, which left an indelible mark on all his future outlook on life, Schumann attempted to commit suicide by throwing himself out of the high window of his room. In the years that followed, there were periods of apparently complete recovery, followed by relapses which gradually became more severe and prolonged. The symptoms were growing anxiety, moodiness and depression, odd manners, tendency to remain silent, suspicion, persistent auditory hallucinations, slow, difficult speech. The progress of the disease was slow, remittent, but inexorable, ending in profound melancholia. At his own request, Schumann was taken to an asylum. There his mental and physical strength gradually declined, and he died peacefully at the age of 51.

It has been assumed that the cause of his death was general paralysis. The writer thinks, however, that this history points without doubt to a diagnosis of dementia præcox. One can hardly say of a man who in the course of an illness extending over

twenty years showed himself a master, not merely as the creator of exquisite music, but as a writer, a poet, and a critic, who was an excellent husband, father, and friend, that he was an imbecile. But dementia præcox numbers among its victims many who never come under this designation. Not a few cases of the nervous weakness of youth belong to this class; after a few years, recovery seems to have taken place, but a subtle change has been left which may be evident only to the sharpest eye. Some are recognised as being weak-minded; in others the mental power seems to be unaltered, but they have lost the gaiety and spontaneity of youth, their ideals have gone, they are no longer capable of passion, and are always either tired or very easily fatigued. Between these slight forms and the severe cases found in the asylums, there are many transition stages, and to these Schumann's case belongs.

By those around him, Schumann's illness was attributed to over-work and mental strain. But no mental disease, whether dementia præcox or paralysis, is caused by mental strain. It is possible that over-excitement favours the onset of the illness, and certain that it has a very prejudicial effect on a disease already established; but the main or only cause of mental disease is a certain inherited disposition—an abnormally constituted brain. Creative work naturally entails fatigue. But if one is tolerably healthy, the fatigue passes off without harm; if one is ill, it easily leads to exhaustion. There is now a prevailing tendency to trace every disease to some early experience, and if fatigue aggravates a disturbance, to regard it as the cause. Schumann worked willingly and joyfully. In his later years he bore work badly, because he was ill; he was not ill because he worked.

The writer therefore concludes that Schumann's disease was dementia præcox, caused by inherited predisposition, and that there is no evidence pointing to the existence of general paralysis.

**THE JOURNAL OF ABNORMAL PSYCHOLOGY.** Editor: MORTON PRINCE, M.D. Vol. i., No. 1, April 1906. The Old Corner Book Store, Inc., Boston, Mass.

**THERE** is no doubt that there is room at the present moment for a journal to represent that department of mental science which the new bi-monthly, *Journal of Abnormal Psychology*, proposes to cover. In the first number the scope of the journal is defined as being "primarily intended for the publication of articles embodying clinical and laboratory researches in abnormal mental phenomena. . . ." The field of investigation includes, for instance, such subjects as hysteria, hallucinations, delusions, amnesias, abulias, aphasias,

fixed ideas, obsessions, deliria, perversions, emotions and their influence, exaltations, depressions, habit neuroses and psychoses, phenomena of hypnosis, sleep, dreams, automatisms, alterations of personality, multiple personality, dissociation of consciousness, sub-conscious phenomena, relation of the mind to physiological processes, neurasthenic and psychasthenic states.

The journal intends to publish the results of clinical and laboratory analyses of the phenomena referred to, rather than mere reports of psychiatric cases. Such analyses should be extremely helpful and suggestive to psychologists and to those working at the problems of clinical psychiatry, and should offer valuable hints in the direction of lines of clinical research. The names of the associate editors, of whom there are six, warrant the assumption that the journal will be broad in its interests, and representative of the various aspects under which these phenomena may be considered. The first number is one of much interest, and promises well for the future of the journal. Abstracts from the articles in the first number are given in the June number of the *Review*.

C. MACFIE CAMPBELL.

**GEHIRN UND SEELE.** Vorlesungen von Prof. Dr. med. PAUL SCHULTZ, Univ. Berlin. Edited by Dr HERMANN BEYER. Leipzig: J. A. Barth. 1906. Pp. 189.

At the end of last July, Prof. Schultz died from heart failure, in the midst of a life full of activity and widespread interests. He had contemplated for some time publishing these lectures in book form, but, finding this impossible, he entrusted the task of their publication to his friend Dr Beyer. The fifteen lectures which comprise the book make no pretence of original investigation or conception, are treated in popular scientific fashion, and are of metaphysical rather than psychiatric interest. This book, whose author was thoroughly imbued with Kantian idealism, is essentially a protest against mechanistic theories of the "soul," and in particular a somewhat belated refutation of what Prof. Lodge has called the "rudimentary, antiquated, gratuitous, hypothetical, in some places erroneous and altogether unconvincing" scheme of materialistic monism formulated by Haeckel. The first three lectures are largely historical; the succeeding six are concerned with the phylogenetic development of the nervous system, and the gradual elaboration and increasing complexity of consciousness and intelligence; and the remaining chapters treat, in rather superficial and discursive fashion, of the relation of brain weight to intelligence, cortical localisation, insanity, hallucinations and illusions,

and hypnosis. To sum up the main argument, mental processes are not spatial phenomena localised in the brain: brain and mind are related only *in time* as parallel psychic processes, and cannot be causally connected, any reciprocal action between the two series being invalid, and mutually inexplicable—i.e. the nature of mind cannot be elucidated by an explanation of the life of the organism, as it can never be the object of exact scientific investigation.

The author is thus an orthodox exponent of psycho-physical parallelism, and though the work is largely outside the field of psychiatry, it is throughout of considerable philosophic interest.

R. CUNYNGHAM BROWN.

---

# Bibliography

## ANATOMY

- PFLÜGER. Ueber den elementaren Bau des Nervensystems. Martin Hager. Bonn, 1906, M. 3.
- TROLARD. Le faisceau longitudinal inférieur du cerveau. *Rev. Neurol.*, mai 30, 1906, p. 440.
- LASALLE ARCHAMBAULT. Le faisceau longitudinal inférieur et le faisceau optique central (suite et fin). *Nouv. Icon. de la Salpêtrière.*, mars-avril 1906, p. 178.
- SERGIO SERGI. Über den Verlauf der centralen Bahnen des Hypoglossus im Bulbus. *Neurol. Centralbl.*, Juni 16, 1906, S. 550.
- W. v. BECHTEREW. Über die absteigenden Verbindungen des Thalamus. *Neurol. Centralbl.*, Juni 16, 1906, S. 546.
- WEIGNER. Über das Hirngewicht des Menschen. *Archiv. f. Anat. u. Physiol.*, 1906, p. 195.
- REICHARDT. Über das Gewicht des menschlichen Kleinhirnes im gesunden und kranken Zustande. *Allg. Zeit. f. Psychiat.*, 1906, p. 183.
- F. T. LEWIS. The Mixed Cerebral Nerves in Mammals. *Journ. of Comp. Neurol. and Psychol.*, May 1906, p. 177.
- DEXLER and MARGULIEZ. Über die Pyramidenbahn des Schafes und der Ziege. *Gegenbaurs morpholog. Jahrbuch*, Bd. 53, p. 413.
- BRODMANN. Über den allgemeinen Bauplan des Cortex pallii bei den Mammaliern und zwei homologe Rindenfehler im besonderen. Barth, Leipzig, 1906, M. 9.
- W. M. SMALLWOOD. Preliminary Report on the Cytology of Molluscan Nerve Cells. *Journ. Comp. Neurol. and Psychol.*, May 1906, p. 183.

## PHYSIOLOGY

- MUNK. Über die Functionen des Kleinhirns. Reimer, Berlin, 1906, M. 2.
- AUERBACH. Beitrag zur Lokalisation des musikalischen Talentos im Gehirn und am Schädel. *Archiv. f. Anat. u. Physiol.*, 1906, p. 197.
- REIGNER. Beiträge zur Physiologie der Kieferbewegungen. *Archiv. f. Anat. u. Physiol.*, 1906, p. 109.
- F. S. LEE. Fatigue. *Journ. of Amer. Med. Assoc.*, May 19, 1906, p. 1491.
- ZOTH. Über die Form der Arbeit am Masso'schen Ergographen. *Archiv. f. d. gesamte Physiologie*, Bd. 112, H. 7 u. 8, p. 311.
- BING. Experimentelles zur Physiologie der Tractus spino-cerebellarea. *Archiv. f. Anat. u. Physiol.*, 1906, p. 250.
- VELICH. Studien über den Einfluss des Nervensystems auf den Puls. *Wien. klin. Woch.*, Mai 31, 1906, p. 663.
- DAVID FRASER HARRIS and WILLIAM MOODIE. On the Non-Uniformity in the Rate of Discharge of Impulses from Cells of the Spinal Cord poisoned with Strychnine. *Journ. Physiol.*, May 31, 1906, p. 213.
- GUIDO GUERRINI. Di una proprietà meccanica del muscolo che si può chiamare "potenza." *Sperimentale*, Anno lx., f. iii., 1906, p. 415.

## PSYCHOLOGY

- ALSBERG. Die Grundlagen des Gedächtnisses, der Vererbung und der Instinkte. Reinhardt, München, 1906, M. 1.
- BAUGH. The Origin of Human Mind. *Canadian Practitioner and Review*, May 1906, p. 239.
- CLAPARÈDE. The Value of Biological Interpretation for Abnormal Psychology. *Journ. of Abnormal Psychology*, June 1906, p. 83.
- PACKARD. The Feeling of Unreality. *Journ. of Abnormal Psychology*, June 1906, p. 69.
- WILIBALD A. NAGEL. Observations on the Colour-Sense of a Child. *Journ. Comp. Neurol. and Psychol.*, May 1906, p. 217.
- PFLAUM. Die individuelle und die soziale Seite des seelischen Lebens. Bergmann, Wiesbaden, 1906, M. 1.60.
- BUSCHAN. Gehirn und Kultur. Bergmann, Wiesbaden, 1906, M. 1.20.
- The Psychology of the Anarchist. Leading Article. *Brit. Med. Journ.*, June 6, 1906, p. 1365.
- CLAYE SHAW. A Lecture on Mind and Matter. *Brit. Med. Journ.*, June 9, 1906, p. 1335.
- RAYMOND PEARL. On the Correlation between Intelligence and the Size of the Head. *Journ. Comp. Neurol. and Psychol.*, May 1906, p. 189.
- ROBERT M. YERKES. George Bohn's Studies in Animal Behaviour. *Journ. Comp. Neurol. and Psychol.*, May 1906, p. 231.
- S. J. HOLMES. Death-Feigning in Ranatra. *Journ. Comp. Neurol. and Psychol.*, May 1906, p. 205.

## PATHOLOGY

- ARMAND-DELILLE. Lésions Nerveuses cellulaires produites par le Sérum névro-toxique. (Soc. de neurol.) *Rev. Neurol.*, mai 30, 1906, p. 469.
- CATOLA und ACHÚCARRO. Über die Entstehung der Amyloidkörperchen im Zentralnervensystem. *Virchow's Archiv.*, Bd. 184, H. 3, p. 454.
- MEDEA. Recherches expérimentales sur la Dégénération et la Régénération des Fibres Nerveuses dans la Névrite parenchymateuse dégénérative. (Soc. de neurol.) *Rev. Neurol.*, mai 30, 1906, p. 483.
- LUGARO. Osservazioni sui "gomitoli" nervosi nella rigenerazione dei nervi. *Riv. di Patol. nerv. e ment.*, vol. xi., f. 4, 1906, p. 170.
- PIERCE CLERK. Do Central Tracts of the Nervous System Regenerate? *New York Med. Journ.*, June 2, 1906, p. 1116.
- GUIDO GUERRINI. Sulla funzione dei muscoli degenerati. *Sperimentale*, Anno lx., f. iii., 1906, p. 427.
- ASCH. Zur Hypertrophie der quergestreiften Muskeln, speziell des Herzmuskels. Springer, Berlin, 1906, M. 1.20.
- CATOLA. Contributo allo studio dell' anatomia patologica della Malattia di Parkinson. *Riv. di Patolog. nerv. e ment.*, vol. xi., f. 4, 1906, p. 145.
- SPIELMEYER. Zur anatomischen Differential-diagnose der progressiven Paralyse. *Centralbl. f. Nervenheilk. u. Psychiat.*, Juni 1, 1906, S. 425.
- OSKAR FISCHER. Ueber einen eigenartigen Markfasernschwund in der Hirnrinde bei Paralyse. *Wien. klin. Woch.*, Mai 31, 1906, p. 661.

## CLINICAL NEUROLOGY AND PSYCHIATRY

## GENERAL—

- BENEDIKT. Zur Theorie der typischen Degenerationskrankheiten des Nervensystem. *Deutsch med. Woch.*, Juni 26, 1906, p. 1000.
- VON LEUPOLDT. Die Untersuchung von Unfallnervenkranken mit psychophysischen Methoden. *Klinik f. psych. u. nerv. Krankh.*, Bd. 1, H. 2, 1906, S. 130.



WALLACE WOOD. Cerebral Segmentation: a New Method of Reading the Brain. *Med. Rec.*, June 2, 1906, p. 878.

REINHARDT. Ärztliche Nervenanalyse (Psychoanalyse). Konegen, Leipzig, 1906, M. 1.

FRANZ FONCK. Radium und Nervensystem. Georg Thieme, Leipzig, 1906.

#### MUSCLES—

RIEGER. Untersuchungen über Muskelzustände. Fischer, Jena, 1906, M. 2.

NOICA. Deux frères atteints de myopathie progressive. *Nouv. Icon. de la Salpêtrière*, mars-avril 1906, p. 151.

#### PERIPHERAL NERVES—

FREDERICK T. LEWIS. The Mixed Cerebral Nerves in Mammals. *Journ. Comp. Neurol. and Psychol.*, May 1906, p. 177.

GELLY. Les troubles nerveux périphériques au début de la tuberculose pulmonaire (thèse). Firmin, Montane et Sicardi, Montpellier, 1906.

JOSEPH STARK. Neuritis in Phthisis. *Brit. Med. Journ.*, June 23, 1906, p. 1464.

RUDLER. Un cas de neurofibromatose généralisée. *Nouv. Icon. de la Salpêtrière* mars-avril 1906, p. 161.

HAMMERSCHLAG. Ein Fall von neurofibromatose Recklinghausen'sche Krankheit mit Beteiligung des Gehörorganes. *Monatsschrift f. Ohrenheilk.*, 1906, p. 309.

#### SPINAL CORD—

**Tabes.**—SIR WILLIAM GOWERS. A Lecture on the Dystrophy of Tabes and the Problem of Trophic Nerves. *Brit. Med. Journ.*, June 2, 1906, p. 1267.

MOUTOT. Sur la coexistence des lésions syphilitiques tertiaires avec le tabes. Gainche, Paris, 1906.

DEBOVE. Tabes et aneurysme aortique. *Journ. de Practiciens*, June 9, 1906, p. 353.

DE BORD YOUNG. Diagnosis of Tabes in the Preataxic Stage, *New York Med. Journ.*, June 2, 1906, p. 1131.

VON RAITZ. The Treatment of Tabes Dorsalis. *Med. Record*, May 19, 1906, p. 786.

**Poliomyelitis Anterior Acuta.**—HUET et LEJONNE. Deux cas de Poliomyélite antérieure aiguë de l'adulte. (Soc. de neurol.) *Rev. Neurol.*, mai 30, 1906, p. 474.

**Progressive Muscular Atrophy.**—DE BUCK et DEROUBAIX. Notes sur un cas d'atrophie musculaire progressive. *Journ. de Neurol.*, mai 5, 1906, p. 161.

**Amyotrophic Lateral Sclerosis.**—LEJONNE et LHERMITTE. Un cas de Sclérose Amyotrophique à forme anormale avec autopsie. (Soc. de neurol.) *Rev. Neurol.*, mai 30, 1906, p. 485.

ROSSI et ROUSSY. Un cas de sclérose latérale amyotrophique avec dégénération de la voie pyramidale suivie au Marchi de la moelle jusqu'au cortex. *Rev. Neurol.*, mai 16, 1906, p. 393.

**Myelitis.**—W. B. WARRINGTON and JOHN OWEN. The Pathology of Myelitis Acutissima Hæmorrhagica Disseminata. *Rev. Neurol. and Psychiat.*, June 1906, p. 401.

ROSENBEGGER u. SCHMINCKE. Zur Pathologie der toxischen Graviditätsmyelitis. *Virchows Archiv.*, Bd. 184, H. 3, p. 329.

**Syringomyelia.**—ALQUIER et GUILLAIN. Études anatomocliniques d'un cas de syringomyélie spasmodique. *Rev. Neurol.*, juin 15, 1906, p. 489.

**Tumour.**—H. OPPENHEIM u. M. BORCHARDT. Ueber zwei Fälle von erfolgreich operierter Rückenmarkshautgeschwulst. *Berlin. klin. Woch.*, Juni 23, 1906, p. 864.

## BRAIN—

- Meningitis.**—GHON, MUCHA, and R. MULLER. Zur Aetiologie der akuten Meningitis. *Centralbl. f. Bakt., etc.* Bd. xli., H. 4, p. 401. Fortsetzung folgt.
- F. KRAUSE. Zur Kenntniss der Meningitis serosa spinalis. *Berlin. klin. Woch.*, Juni 18, 1906, p. 817.
- JEHLE. Ueber das Entstehen der Genickstarrepidemie. *Wien. klin. Woch.*, Juni 21, 1906, p. 753.
- J. S. BILLINGS, Jr. Cerebro-spinal Meningitis in New York City during 1904-05. *Journ. of Amer. Med. Assoc.*, June 2, 1906, p. 1670.
- HYBRAM. Contribution à l'étude de la méningite cérébro-spinale. Relation d'une épidémie dans la province d'Oran (Algérie). Privat, Toulouse, 1906.
- HUNTER. Note on an Atypical Case of Cerebro-spinal Meningitis. *Lancet*, May 26, 1906, p. 1469.
- GILLARD. Méningite tuberculeuse au cours d'une granulée polynucléose rachidienne. *Arch. gén. de méd.*, juin 5, 1906, p. 1421.
- E. SCHLESINGER. Der therapeutische und symptomatische Wert der Lumbalpunktion bei der tuberkulösen Meningitis der Kinder. *Berlin. klin. Woch.*, Juni 18, 1906, p. 838.
- Hæmorrhage.**—HERBERT J. WALKER. A Case of Meningeal Hæmorrhage resembling Hysteria. *Lancet*, May 26, 1906, p. 1468.
- W. B. WARRINGTON and JOHN OWEN. A Case of Hæmorrhage into the Brain and Spinal Cord from Obliterative Arterial Disease. *Rev. Neurol. and Psychiat.*, June 1906, p. 407.
- Hemiplegia.**—H. G. LANGWILL. Transitory Hemiplegia, with Notes of Two Cases. *Scot. Med. and Sur. Journ.*, June, 1906, p. 509.
- Tumour.**—PUTNAM and WATERMAN. A Contribution to the Study of Cerebellar Tumours and their Treatment. *Journ. Nerv. and Ment. Dis.*, May 1906, p. 297.
- RAYMOND. Un cas de tumeur du cervelet. *Journ. der Practiciens*, juin 2, 1906, p. 337.
- E. E. SOUTHARD. Case of Glioma of the Frontal Lobe, with Invasion of the Opposite Hemisphere. *Amer. Journ. Insan.*, April 1906, p. 561.
- CESTAN. Epithélioma primitif du Cerveau. (Soc. de neurol.) *Rev. Neurol.*, mai 30, 1906, p. 468.
- PÜSCHMANN. Fall von Kleinhirnbrückengeschwulst. *Deutsch med. Woch.*, Mai 24, 1906, p. 836.
- ALQUIER. Volumineux tubercule caséifié de la calotte protubérantielle. Étude anatomo-clinique. *Rev. Neurol.*, mai 15, 1906, p. 406.
- HUGH SMITH and C. C. ELLIOTT. A Case of Cerebral Tumour; Operation; Recovery. *Lancet*, June 16, 1906, p. 1688.
- Cerebral Diplegia.**—DANNENBERGER. Über die porenkephalische Form der zerebralen Kinderlähmung. *Klinik f. psych. u. nerv. Krankh.*, Bd. 1, H. 2, 1906, S. 100.
- General Paralysis.**—VORBERG. Dementia paralytica und Syphilis. Deuticke, Wien, M. 1.
- PLASKUDA. Ein Fall von progressive Paralyse mit gehäuften epileptiformen Krämpfen, etc. *Allg. Zeit. f. Psychiat.*, 1906, p. 240.
- RAMSAY HUNT. Chronic Progressive Softening of the Brain. *Amer. Journ. of the Med. Sci.*, June 1906, p. 1020.

## MENTAL DISEASES—

- F. W. MOTT. An Address on the Pathological Investigation of the Causation of Insanity. *Lancet*, June 2, 1906, p. 1515.
- SOUTZO FILS. La psychiatrie moderne et l'œuvre du professeur Kraepelin (suite et fin). *Ann. méd.-psychol.*, mai-juin 1906, p. 402.
- CLARENCE B. FARRER. Clinical Psychiatry. *Amer. Journ. of Insan.*, April 1906, p. 627.
- PENTA. Die Simulation von Geisteskrankheit. *Stubers Verlag*, Würzburg, 1906, M. 3.50.
- VICTOR PARANT. Un article de M. le professeur Grasset: "Demi-fous et demi-responsables." *Ann. méd.-psychol.*, mai-juin 1906, p. 353.

- PHILIPPE et PAUL-BONCOUR. Les Anomalies Mentales chez les Écoliers. Félix Alcan, Paris, 1906, 2 fr. 50c.
- ÖFFENHEIMER. Die medizinische Psychologie mit Bezug auf Behandlung und Erziehung der angeboren Schwachsinnigen. *Munch. med. Woch.*, Mai 22, 1906, p. 1023.
- C. J. ROBERTSON-MILNE. Notes on Insanity with Illustrative Cases. *Indian Med. Gaz.*, April 1906.
- JOSEF BERZE. Das Primärsymptom der Paranoia. *Centralbl. f. Nervenheilk. u. Psychiat.*, Juni 1, 1906, S. 432.
- C. W. HITCHCOCK. A Case of Dementia Præcox of Medico-Legal Interest. *Amer. Journ. of Insan.*, April 1906, p. 615.
- F. X. DERCUM. The Heboid-Paranoid Group (Dementia Præcox). Clinical Relations and Nature. *Amer. Journ. Insan.*, April 1906, p. 541.
- HERZER. Beitrag zur Klinik der Puerperalpsychosen. *Allg. Zeit. f. Psychiat.*, 1906, p. 244.
- ALBRAND. Das psychische Verhalten von Geisteskranken im Sterben. *Allg. Zeit. f. Psychiat.*, 1906, p. 299.
- MAURICE DIDE et ALBERT LEBORGNE. Symptômes et Lésions Médullaires dans la Démence Précoce catatonique. (Soc. de neurol.) *Rev. Neurol.*, mai 30, 1906, p. 479.
- BEURMANN, ROUBINOVITCH et GOUGEROT. Les Troubles mentaux dans la lèpre. Gainche, Paris, 1906.
- KÖLPIN. Die psychischen Störungen nach Kopftraumen. Breitkopf & Härtel, Leipzig, 1906, M. 0.75.
- MARANDON de MONTYEL. Les Pervers. *Journ. de Neurol.*, mai 20 et juin 5, 1906, pp. 181, 201.
- TOURENC. État Mental des Incendiaires. Michalon, Paris, 1906, 2 fr. 50 c.
- GIMBAL. Les incendiaires (suite) *Ann. méd.-psychol.*, mai-juin 1906 p. 380.
- ROUSSET. Un cas d'exhibitioniste. *Ann. méd.-psychol.*, mai-juin 1906, p. 394.
- T. CLAYE SHAW. Insanity and Murder. *Lancet*, June 23, 1906, p. 1739.
- NOUËT. De la Necessité d'exiger des Connaissances Psychiatriques pour les Médecins des Établissements Pénitentiaires. Michalon, Paris, 1906, 3 fr.
- MOELI. Die in Preussen gültigen Bestimmungen über die Entlassung aus den Anstalten für Geisteskranke. Marhold, Halle, 1906, M. 1.20.

#### ALCOHOL—

- ISADOR H. CORIAT. The Mental Disturbances of Alcoholic Neuritis. *Amer. Journ. of Insan.*, April 1906, p. 571.
- DANNEMANN. Über Bewusstseinsveränderungen und Bewegungstörung durch Alkohol, besonders bei Nervösen. *Klinik. f. psychis. u. nerv. Krankh.*, Bd. 1, H. 2, 1906, S. 79.

#### SPECIAL SENSES AND CRANIAL NERVES—

- PICK. Über Hyperästhesie der peripherischen Gesichtsfeldpartien. *Neurol. Centralbl.*, Juni 1, 1906, S. 498.
- BRAILLON. Des réflexes pupillaires dans les cardiopathies mitrales. *Gaz. des Hôp.*, juin 21, 1906.
- NEUMANN. Die otitischen Facialisparesen. *Wien. med. Woch.*, Juni 16 u. 23, 1906, pp. 1234 u. 1306.
- MIRALLIÉ et PLANTARD. Paralysie Faciale double au cours d'une Polynévrite infectieuse généralisée. (Soc. de neurol.) *Rev. Neurol.*, mai 30, 1906, p. 480.
- MASSEL. Un segno premuntorio della paralisi del ricorrente laringeo. *Arch. ital. di laringol.*, F. 1, 1906.

#### GENERAL AND FUNCTIONAL DISEASES—

- Chorea.**—TRONIBETTA. Lo strapazzo intellettuale e le nevrosi (contributo all' etiologia della corea). *La Clinica Moderna*, 11 aprile 1906, p. 169.
- Sir DYCE DUCKWORTH. Remarks on Chorea considered as Cerebral Rheumatism. *Brit. Med. Journ.*, June 23, 1906, p. 1464.
- Epilepsy.**—REDLICH. Bemerkungen zur Ätiologie der Epilepsie. *Wien. med. Woch.*, Mai 26 u. Juni 2, 1906, p. 1074 u. 1147.

- Neurasthenia.**—MÖBIUS. Die Nervosität. Weber, Leipzig, 1906, M. 2.50.  
 ALDER BLUMER. The Coming of Psychasthenia. *Journ. Nerv. and Ment. Dis.*, May 1906, p. 336.  
 DONLEVY. On Neurasthenia as a Disintegration of Personality. *Journ. of Abnormal Psychol.*, June 1906, p. 55.  
**Hysteria.**—HAASE. Über eine Epidemie von hysterischem Laryngismus. *Wien. med. Presse*, Juni 3, 1906, p. 1187.  
 ARSIMOLES. Hystérie infantile avec hallucinations. Gainche, Paris, 1906.  
 PAUL BLUM. Des Anesthésies psychiques dites Nerveuses ou Hystériques. Octave Doin, Paris, 1906, 5 fr.  
**Torticollis.**—A. H. TUBBY. A Clinical Lecture on Torticollis, or Wry-Neck. *Brit. Med. Journ.*, June 16, 1906, p. 1387.  
**Angio-Neurotic Edema.**—TRUMAN. A Case of Angio-Neurotic Edema. *Lancet*, June 2, 1906, p. 1535.

## MISCELLANEOUS SYMPTOMS—

- CLAUDE et LEJONNE. Hypotrophie d'origine bacillaire; troubles de la voie pyramidale. *Nouv. Icon. de la Salpêtrière*, mars-avril 1906, p. 147.  
 ROEDER. Zwei Fälle von linksseitiger Abduzenslähmung nach Rückenmarksanästhesie. *Münch. med. Woch.*, Juni 5, 1906, p. 1118.  
 KÖNIG. Bleibende Rückenmarks-lähmung nach Lumbal-Anästhesie. *Münch. med. Woch.*, Juni 5, 1906, p. 1112.  
 OTFRID FÖRSTER. Die Kontrakturen bei den Erkrankungen der Pyramidenbahn. Karger, Berlin, 1906, M. 1.60.  
 JULES VOISIN, ROGER VOISIN et A. RENDU. Idiotie et lésion cérébelleuse. Amélioration des symptômes. *Arch. gén. de méd.*, mai 29, 1906, p. 1865.  
 ALFRED GORDON. Atrophy of the Intrinsic Muscles of the Hands due to Lead Poisoning. *New York Med. Journ.*, June 2, 1906, p. 1125.  
 MORITZ. Mitbeteiligung des Phrenicus bei Duchenne-Erb'scher Lähmung. *Deutsch. med. Woch.*, Juni 7, 1906, p. 909.  
 SOCA. Sur un cas de "paralyse de bequilles." *Nouv. Icon. de la Salpêtrière*, mars-avril 1906, p. 171.  
 A. PICK. Bemerkungen zur Pathologie der Akroparästhesie. *Berlin. klin. Woch.*, Juni 4, 1906, p. 745.  
 HENSCHEN. Zum bulbären Syndrom: Dissoziation der Sinne in Verbindung mit cerebellar-ataktischen Störungen. *Neurol. Centralbl.*, Juni 1, 1906, S. 502.  
 GRASSET. La claudication intermittente des centres nerveux. *Rev. Neurol.*, mai 30, 1906, p. 433.  
 LEJONNE et EGGER. Traumatisme crânien. Syndrome vestibulaire. Accidents méningés cérébro-spinaux. (Soc. de neurol.) *Rev. Neurol.*, mai 30, 1906, p. 470.  
 ANGELL. Hypæsthesia and Hypalgesia and their Significance in Functional Nervous Disturbances. *Journ. Nerv. and Ment. Dis.*, May 1906, p. 324.  
 PFISTER. Zur Ätiologie und Symptomatologie der Katatonie. *Allg. Zeit. f. Psychiat.*, 1906, p. 275.  
 FEILER. Über zwei instructive Fälle von Sympathicusneurose und über ein bei denselben aufgetretenes anfallendes Symptom. *Wien. med. woch.*, Juni 2, 1906, p. 1130.  
 BRUMPT. Doigts en Lorgnette au cours d'une Atrophie Musculaire Progressive chez un nègre du Soudan. (Soc. de neurol.) *Rev. Neurol.*, mai 30, 1906, p. 477.  
 HANS KAHN. Studien über den Schluckreflex. *Arch. f. Anat. u. Physiol.*, 1906, p. 355.  
 JAMES MACKENZIE. Remarks on the Meaning and Mechanism of Visceral Pain as shown by the Study of Visceral and other Sympathetic (Autonomic) Reflexes. *Brit. Med. Journ.*, June 23, 1906, p. 1449.  
 A. PICK. Ueber motionisch-bedingte Mikrographie. *Wien. klin. Woch.*, Juni 21, 1906, p. 757.  
 HEILBRÖNNER. Sprachstörungen bei funktionellen Psychosen mit Ausschluss der aphasischen Störungen. *Centralbl. f. Nervenheilk. u. Psychiat.*, Juni 15, 1906, S. 465.  
 BYROM BRAMWELL. A Series of Lectures on Aphasia. Lecture III. *Lancet*, June 16, 1906, p. 1671.

## TREATMENT—

- A. T. SCHOFIELD. The Management of a Nerve Patient. J. & A. Churchill, London, 1906. 5s.
- BRÜGELMANN. Die Behandlung von Kranken durch Suggestion und die wahre wissenschaftliche Bedeutung derselben. Eine psychiatrischen Studie. Thieme, Leipzig, 1906, M. 1.20.
- ERTL. Vollständiger Lehrkurs des Hypnotismus in allen seinen Phasen und verwandten Erscheinungen. Fiedler, Leipzig, 1906, M. 2.
- MATHIEU et ROUX. Traitement de l'hystérie gastrique. *Gaz. des Hôp.*, juin 12, 1906.
- POROT. Le traitement arsenical de chorée. *Gaz. des Hôp.*, juin 2, 1906.
- ALT. Ernährungstherapie der Basedow'schen Krankheit. *Münch. med. Woch.*, Juni 12, 1906, p. 1145.
- GERLACH. Versuche mit Neuronel bei Geisteskranken. *Münch. med. Woch.*, mai 22, 1906, p. 1017.
- LEHMANN. Essai sur l'action thérapeutique du radium. *Arch. gén. de méd.*, mai 22, 1906, p. 1301.
- ANTONIN CONVERS. De l'action de l'acide formique en médecine mentale. *Ann. méd.-psychol.*, mai-juin 1906, p. 388.
- ESCHERICH. Die Verwendung der Pyozyanese bei der Behandlung der epidemische Säuglingsgrippe und der Meningitis cerebrospinalis. *Wien. klin. Woch.*, Juni 21, 1906, p. 751.
- OSTWALT. On Deep Alcohol Injections in Facial and other Neuralgias and in Histrionic Spasm. *Lancet*, June 9, 1906, p. 1605.
- WILENS. Heilung hysterischer Kontrakturen durch Lumbalähmung. *Deutsch. med. Woch.*, Juni 14, 1906, 954.
- SCOTT CARMICHAEL. A Plea for Operative Interference in Intracranial Hæmorrhages in the New-born. *Scot. Med. and Sur. Journ.*, June 1906, p. 524.

## SECOND CONGRÈS BELGE

DE

## NEUROLOGIE ET DE PSYCHIATRIE

(Bruxelles, 29 au 31 août 1906)

## Présidents d'Honneur

M. LE BARON VAN DER BRUGGEN  
Ministre de l'AgricultureM. VAN DEN HEUVEL  
Ministre de la Justice

## Vice-Présidents d'Honneur

M. BÉCO  
Gouverneur de la Province de BrabantM. DE LATOUR  
Directeur général au Ministère de la Justice

## Travaux scientifiques

1<sup>o</sup> RAPPORTS.—Ces rapports, au nombre de trois, seront distribués au moins trois semaines avant la réunion du Congrès, de manière à pouvoir être attentivement étudiés par ceux qui désirent prendre part à la discussion.

Les questions choisies pour la session de 1906 sont :

(a) PSYCHIATRIE : Les aliénés dissimulateurs.—Rapporteurs : M. le Dr DE MOOR, médecin en chef de l'hospice Gulstain, à Gand, et M. le Dr DUCHATEAU, médecin de la Maison de santé pour femmes, à Gand.

(b) NEUROLOGIE : La théorie du neurone.—Rapporteur : Mlle. le Dr STEFANOWSKA, assistante au laboratoire de psychologie de l'Université de Bruxelles.

(c) PSYCHOLOGIE : Les tests mentaux chez les enfants.—Rapporteurs : M. le Dr DECKOLT, directeur de l'Ecole d'enseignement spécial de Bruxelles, et M. le Dr BOULENGER, médecin-adjoint de l'Asile d'aliénés au Fort Jaco à Uccle.

2<sup>o</sup> COMMUNICATIONS DIVERSES.—Une place importante est réservée aux communications originales sur un sujet quelconque de neurologie ou de psychiatrie, avec présentation de malades, de pièces anatomiques et microscopiques.

Les membres qui désirent faire une communication au Congrès sont priés d'en envoyer le titre et le résumé au secrétaire général avant le 30 juin 1906.

Les séances du Congrès seront combinées avec la visite d'instituts scientifiques et hospitaliers.

Des réductions de prix seront demandées sur les chemins de fer français.

Le prix de la cotisation est de 10 francs. Envoyer les adhésions à M. le Dr MASSAUT, secrétaire général, médecin directeur de la colonie d'aliénés de Lierneux.

Les praticiens de toutes nationalités peuvent faire partie de ce Congrès ; la seule restriction imposée est l'usage d'une des langues usitées en Belgique.

# Review of Neurology and Psychiatry

---

## Original Article

### THE PATHOLOGY OF GENERAL PARALYSIS.

By DR HANS EVENSEN,

Medical Superintendent of Trondhjem Lunatic and Criminal Asylum

(Lecture delivered before the University of Christiania.)

#### PART I.

#### MACROSCOPIC EXAMINATION.

THE changes which may be seen, even with the naked eye, in a post-mortem examination of a case of general paralysis are in general characteristic, and they have led, as we know, to the earliest interpretation of its pathology as a chronic arachnitis (Bayle, 1822), or a chronic diffuse peri-encephalo-meningitis (Calmeil, 1826).

The skull, as a general rule, is thicker and denser than normally, the diploë having given place to compact tissue.

The *dura mater* may adhere more closely to the skull than usual, but the adhesion is seldom so firm as, for instance, in cases of senile insanity. The membrane is thicker and less translucent, but as the thickness varies greatly under normal conditions, it is not easy to pronounce with certainty as to this matter in each case. The inner surface is generally smooth and glossy; it may also be rough, or it may display deposits ranging from a film which is just visible, colourless or reddish, and which

cannot be washed off, to thick, pulpy and highly vascularised newly-formed membranes, which lie in layers on the top of each other and are the seat of hæmorrhage. A very large hæmatoma of the dura mater is very seldom met with ; it was, perhaps, more frequent at one time, when treatment could not protect the patient from injuries in the same degree as now. Occasionally there are only rusty patches to be seen on the inside, and no membrane ; but on the other hand, the dura mater may not display any change whatever. Along the middle line it may be adherent to the pia round the Pacchionian bodies. Occasionally the latter are larger than usual.

The *leptomeninges* (pia-arachnoid)—for I agree with Ford Robertson that it is artificial to distinguish between an exterior alleged non-vascular membrane (arachnoidea) and an inner vascular one (pia)—present a very milky aspect and are somewhat thickened and often distended over the sulci, which are filled with arachnoid fluid. In itself this fluid is as a rule quite clear, but seen through the pia it resembles whey. The exudate is not purulent unless an infection has supervened (most usually pneumonia). The milkiness is specially evident over the convexity of the cerebrum and along the vessels, most of all over the frontal lobe and the front part of the parietal lobe. It may be so dense that one cannot distinguish the outlines of the convolutions. On the inferior surface of the brain, where the changes in the membrane are not on the whole very pronounced, it is most easily seen at the commencement of the Sylvian fissure and between the pons and the cerebellum. The small vessels in the membrane are often gorged with blood, but as it is not always an evidence of the amount of blood supply during life, and may depend on the mode of death, this circumstance is of minor importance. Of greater importance is the fact that over a large extent the pia-arachnoid cannot be detached from the cortex without bringing some of the latter with it, mainly over the summit of the convolutions, and especially when the membrane is not considerably thickened. But here also external circumstances play a part, as the adhesion is more pronounced the more advanced the post-mortem changes are. The membrane is occasionally finely granular. The pole of the occipital lobe is, as a rule, quite free from changes of the soft membrane, and there are cases where the condition of the membranes, on the whole, does

not support the assumption of a general paralysis. In other cases again the occipital lobe is chiefly affected, and it has been thought that this is particularly the case when the disease begins with tabes.

The larger *cerebral arteries*, especially at the base, often appear rigid, widely gaping on being cut across, and dotted over with white or yellow spots. These changes, which in general are looked upon as a sign of senility, setting in at the ordinary age or prematurely, are not confined to general paralysis. Further, by cutting across an apparently normal artery one can sometimes, even with the naked eye, distinguish a fairly uniform thickening of the inner coat of the artery extending round a greater or smaller portion of the transverse section, but as a rule it cannot be decided by the unaided vision whether the vessel wall is altered or not.

The *brain matter* itself is generally of somewhat softer consistence than normally; it is seldom firmer. The cortex may be narrowed and its details effaced, being of a uniform reddish-grey colour or spotted with red. There is, however, so much room for a subjective estimate in forming an opinion as to the macroscopic appearance, that its practical value is comparatively small. As long as writers confined themselves to recording the macroscopic results, these conditions had greater attention devoted to them, and it was quite a common occurrence for the alienist, who was more accustomed to notice minute differences, to think that he saw distinct changes where the general pathologist found nothing to remark. On the line between the cortex and the white matter, and in the white matter itself, there are sometimes to be seen pale stripes or spots corresponding to the loss of bands of medullated fibres (Tuczek), and it has been thought that in these stripes we have a certain macroscopic sign of general paralysis. In cases of longer duration the convolutions are distinctly narrowed, especially in the front half of the brain, and on being weighed the brain shows a loss of weight up to several hundred grammes. This is the case mainly in regard to the white matter, less so as regards the grey matter. In the atypical forms especially, the thalami optici are often atrophic.

The shrinkage is also seen in the large increase of *arachnoidal fluid* under the soft membrane, but mainly in the ventricles. The lateral ventricles are especially enlarged. The walls of these, as



well as the floor of the fourth ventricle, are not smooth, but feel like sand to the touch, at least in some places, or are covered with small translucent granulations. In the grey matter subjacent to the central ventricles there may occasionally be seen blood effusions, which have set in immediately before death, especially when this has occurred during a paralytic seizure.

Also in other parts of the brain one may meet with slight hæmorrhages or softenings which are visible to the naked eye, but these are not specially frequent even in cases where there is a clinical history of apoplectic attacks. They may, of course, be a consequence of vascular changes which have no direct connection with the paralytic process.

Into the question of the state of the spinal cord in paralysis, especially the common degeneration of the fibre-systems, there will not be time to enter on the present occasion; neither shall I treat of the lesions of the other organs, with the exception of certain points, to which I shall return later.

The macroscopic picture which is generally seen in cases of paralysis is not sufficient evidence of this disease, and it may be absent. The occipital lobe may be most seriously affected; when in that case paralytic seizures with residual focal symptoms are the most prominent in the clinical picture, while the mental decay is less obvious, and increases by fits and starts, and not by a steady progression, then we have the form to which Lissauer gave the name of "atypical paralysis." There are also, however, other deviations from the type in which the macroscopic changes are chiefly restricted to particular regions (the cerebellum, temporal lobe, etc.).

#### MICROSCOPICAL EXAMINATION,

therefore, can alone decide the anatomical diagnosis.

Like a red thread through the history of the pathology of paralysis runs the tendency to class the alterations under the heading of a simple morbid process. At one time the contest centred round the question of primary or secondary alteration, at another the shibboleth was either degeneration or inflammation, and this inflammation again was regarded either as parenchymatous or interstitial. The changes especially which the inflammatory theories underwent during the last century are reflected in the opinions on the pathology of general paralysis.

The disagreement begins even with regard to the membranes. It may be said that the changes here have been the object of less interest, inasmuch as it has generally been taken for granted, ever since the time of Bayle, that these throughout display the ordinary symptoms of a simple chronic inflammation. It is principally

*The subdural newly-formed membranes*

round which the battle has raged. As early as 1826, Bayle and Calmeil saw in them the issue of a pachymeningitis, an assumption which is generally attached to the name of Virchow (1856). Similarly, Huguenin (1877) stands as champion of the opinion which was first put forward by Prescott Hewitt (1845), that the primary change was a hæmorrhage. According to Ford Robertson, who has revived the old question, the inflammatory theory does not hold good. Several of the newly-formed membranes are the result of hæmorrhage in the subdural space, most usually from a pial vein, without any change in the dura itself. It is the vessels from the dura mater alone which make their way into the blood clot, since the surface of the dura mater has most capillaries. Other new membranes are connected with a widespread morbid process in the dura mater, characterised by proliferation and degenerative changes of the surface epithelium, increase of connective tissue fibres, compression of the vessels, and most of all by the formation of new capillaries, numbers of which become obliterated and undergo hyaline changes, partly also fatty degenerations. In the last case they are easily ruptured, and the new membrane is generally developed here also from hæmorrhages, which are then numerous and punctiform. These, however, are not a necessary condition. A somewhat similar chronic morbid alteration in the dura, which cannot rightly be called inflammation, occurs also in cases other than insanity.

The majority of the newly-formed membranes give the impression of comparatively recent formation; some can scarcely be called membranes at all, but consist for the most part of fibrine. If the membrane is organised, there are found, according to Ford Robertson, numerous granules of amorphous hæmatoidin. Mendel, on the other hand, seldom saw debris from disintegration of red corpuscles, and founded on this fact the assumption that the membrane was formed previous to the hæmorrhage.

*In the pia-arachnoid*

also the connective tissue fibres have increased and the endothelial cells on the surface and along the trabeculae show alterations similar to those in the dura. Further, the small vessels are infiltrated (Del Greco, 1891). The new connective tissue fibres, which are specially met with in the superficial layer in patches, or uniformly distributed, are generally coarse, and it is these which cause the thickening of the membrane (Fig. 1). The newly-formed endothelial cells are sometimes accumulated in heaps, and thus the aforementioned small granulations on the surface of the membrane are produced (L. Meyer, 1862). The membrane is also found to be

*Penetrated by numerous smaller cells,*

which have been explained by many as lympho- or leuco-cytes. I am not able to say definitely whether outside the vessel sheaths lymphocytes occur regularly and in greater numbers in the pial tissue in general paralysis, as I have only had the opportunity of seeing a few cases of paralysis since I began systematically to study the membranes. According to Ford Robertson, blood corpuscles are, on the whole, absent. At any rate polynuclear leucocytes will occur only singly, unless septic processes have taken place (Nissl). Even regular occurrences of lymphocytes ought not necessarily to be ascribed to the paralytic process itself; they might as well be due to the preceding syphilitic infection. On the other hand, the absence of white corpuscles does not necessarily exclude inflammation. Those cells which fill the adventitial sheaths and the immediate neighbourhood of the vessels are, as will be proved later on, for the greater part not "round cells," although there are lymphocytes among them. In the brain substance these cells (plasma cells) have undertaken the task which is generally attributed to the leucocytes in inflammations of other regions. Consequently, it ought not to be considered improbable that they should do so in the pia also under similar circumstances. Finally, as far as I can see, some of the cells which lie free in the pial tissue belong to the same group as the infiltrating cells.

After all, perhaps much is not gained by calling the process in the pia by the indefinite name of "inflammation." But when

we take into consideration the condition of the vessels, I suppose we must still maintain the old hypothesis of most authors, that, as a rule, an exudative inflammation occurs in general paralysis. On the other hand, Ford Robertson's opinion is certainly correct that the process is merely hyperplastic in the similar alterations which take place in senile insanity, chronic alcoholism, and epilepsy. Dupré (p. 115) maintains that even here there may be a meningitis.

Among other forms of cells which (in adults) are not normally found in the pia, and which may be found in general paralysis, the "lattice cells" (*Gitterzellen*) (Fig. 2) occur rather frequently, chiefly in areas of blood effusion. They are partly the same cells which older authors called "fatty granular cells" (*Körnchenzellen*) and epithelioid cells. Juliusburger and Boedecker gave them the name of "lattice cells" (*Gitternetzzen*). In the brain they appear diffusely in hæmorrhages and in softenings; they accomplish their task as scavenger cells by taking up and carrying away the debris of medullary substance and disintegrating red corpuscles, and they are temporary substitutes for the destroyed tissue. The "lattice cells are *par excellence* the phagocytic migrating cells of the nervous system" (Nissl).<sup>1</sup> They cannot at all be said to indicate that there is an inflammation.

The cause of the *adhesion* of the pia to the surface of the cortex (decortication) will be found in the condition of the cortex as well as in that of the pia. The pial vessels are more adherent in consequence of the increase of the connective tissue fibres in the adventitia, and of their more intimate connection with the neuroglia. And also the glial fibres along the surface of the convolutions are thicker and more numerous (Weigert), and probably the cortical substance is softer.

Ford Robertson maintains that the cause of the hyperplastic and degenerative alterations of the tissue of the membrane is an altered chemical composition of

*The cerebro-spinal fluid,*

which is said to be effected by the metabolic changes in the brain.

<sup>1</sup> In 1900 Nissl considered these cells which he found in the brain substance as neuroglia cells that had no processes, later on as derived from adventitia (Stroebe), or from endothelial cells. In the pia-arachnoid he thinks they may probably be of autochthonic origin. At all events they are not derived from the blood, as Friedmann maintained. Even what this author called "epithelioid cells," derived from the local tissue cells, were probably chiefly "lattice cells."

From the vessels of the brain the lymph flows through the channels of adventitia into the lymph-spaces of the pia-arachnoidea, and from these into the subdural space and to the dura mater, according to Ford Robertson's view. As yet, however, very little is known about the intracranial lymph circulation. In this connection it is interesting to know that Mott, Donath, and others, by chemical analysis of the cerebro-spinal fluid in general paralysis and other diseases of the brain, have found cholin, a product of decomposed nervous tissue. Since the introduction of lumbar puncture the condition of the cerebro-spinal fluid has been examined with far greater interest than before, and in general paralysis this method of investigation has proved specially valuable. The quantity of albumen will be found to have increased to a much higher degree than in other insanities (Schaeffer, Babcock). This is especially the case with the serum albumen (Guillain et Parant), which is not found in the normal fluid, or at least only in minimal quantities (Sicard). By microscopic examination of the sediment after centrifuging, the whole field under the oil immersion will generally be found overcrowded with lymphocytes, and this result is so constant, and so different from what is seen in other brain diseases, that it has led to the use of lumbar puncture for diagnostic purposes. This was first practised in French clinics. According to Joffroy, lymphocytosis is the earliest certain symptom of general paralysis. But it must not be forgotten that positive reaction may be obtained from an infective meningitic process and from "organic" diseases of the brain, nay even from preceding syphilitic infection, although the number of elements will then be small (Nissl). Polynuclear leucocytes will seldom be found either alone or predominant. Nissl, however, points out that the technical difficulties prevent a more accurate analysis of the elements of the cells, and he maintains that a great number of what seem to be lymphocytes turn out, in successful preparations, to be transitional forms and polymorpho-leucocytes. According to Dupré, the polynuclear leucocytes have migrated from the vessels and they denote a congestion of the membranes, while the lymphocytes come from the lymph sheaths and signify a "simple serous irritation."

The microscopic alterations which in general paralysis take place in

*The intracranial vessels*

(from the arteria basilaris to the smaller pial arteries which dip into the brain substance) are so heterogeneous and vary so much, not only in different cases but in different vessels in the same case, that it is very difficult to deal with them collectively. Only in the cortex itself do the alterations become more alike, and here they show the aspect characteristic of general paralysis. As the conditions are less complicated in the cortex, it is best to commence with it.

One of the first conditions noted by microscopical examination of the cortex in general paralysis was the infiltration of arteries, capillaries, and veins by "small cells." Calmeil was of opinion that the small vessels transmitted the inflammation from the membranes to the brain, and he described the "nucleus-shaped corpuscles" found in the vessel sheaths as "dotted discs" (*disques ponctués*). Later on they were simply called "round cells," and were generally regarded as migrated white corpuscles, although some authors maintained that they originated from the cells of the vessel wall. The staining methods then used did not make it possible to recognise the different forms in the infiltrations, or to distinguish them from the nuclei of the surrounding brain tissue. By careful isolation of what seemed to be nuclei, Ludv. Meyer found (1873) that they were oval cells of the size of white corpuscles, being composed of opaque protoplasm and one large nucleus with one, frequently two, nucleoli. They were not the common cells of the vessel wall. He supposed that by fusion with the vessel wall and by retrogressive changes they transformed the capillaries into homogeneous hyaline tubes. Even the filling up of the lymph-space with nuclei was not considered characteristic of general paralysis alone; in Mendel's monograph (1880) it is said that the same process occurs not only in various diseases of the brain, but even in its healthy state.

As far as I have been able to discover, the first author who traced the real nature of these elements was Oppenheim. In a paper written under his direction (1893), some large cell formations found in a softening in central syphilis are assumed to be plasma cells, a form of cells which had been described by Unna two years before. But possibly Oppenheim meant those plasma cells which were described by Waldeyer in 1875,

and which later on were called by Ehrlich *Mastzellen*. To show the plasma cells in the skin, Unna used the method of hardening in alcohol, staining with methylene blue, and subsequently differentiating. As Nissl's method of staining nerve cells (1894) is based on the same principle, it gave us at the same time the means of distinguishing the plasma cells in the cortex. Alzheimer described and photographed these cells in general paralysis (1897), but Nissl was the first to identify them as plasma cells (1900). In the same year my countryman Ragnar Vogt, under Nissl's direction, undertook to investigate their occurrence. He arrived at the conclusion that the plasma cells were pathognomonic of general paralysis. By further examination of 300 cases, Nissl was able to prove that they were constantly to be found in general paralysis. Yet some authors maintain that they do not occur in many pronounced cases; according to Mahaim they are not found even in 5 per cent. From their description (*e.g.* Havet's), it is however evident that the dissension is owing to their view of what are to be considered as plasma cells. In fact, the reproduction of a drawing which accompanied Vogt's paper, and to which these authors refer, was quite misleading.

By and by it became evident that the terms in which Vogt had summed up his results were too categorical, as plasma cells were also found in brain diseases other than paralysis, as for instance, in non-purulent encephalitis, in rabies, cerebral syphilis, and tuberculous meningo-encephalitis, around abscesses and carcinomatous nodules, and sometimes also in the neighbourhood of softenings. During a visit to the Claybury Laboratory my attention was drawn to their occurrence in the brain, also in the sleeping sickness caused by *trypanosoma Ugandense*, a disease which shows some resemblance to paralysis in other respects also, clinically as well as anatomically (Mott). In a case of arterio-sclerotic insanity I found a few plasma cells among a great number of lymphocytes infiltrating the adventitial lymph space of a large, undoubtedly arterio-sclerotic artery in the cortex. Mahaim found plasma cells even in intoxication from *felix mas*. The fact is generally overlooked that Vogt has also stated, that according to the occurrence of plasma cells in other organs, we must expect to find them in *several* inflammations and inflammatory new-growths; he once found them in

---

meningitis during tuberculosis. The careful reader of his paper will notice that according to Vogt's opinion the plasma cells are not pathognomonic of general paralysis unless they occur diffusely. To prevent a narrow view being taken of the results of examination of the cortex, Nissl has lately found it necessary to emphasise the fact that the plasma cells are only one among other signs of the disease. But this reservation does not impugn the validity of the doctrine—No general paralysis without plasma cells.

Unna defines plasma cells as hypertrophic connective tissue cells in which the granoplasm<sup>1</sup> with amorphous granules has been excessively developed. At the same time, the other constituent of the protoplasm, the reticulated spongioplasm, has diminished from shrinking of the prolongations of the spongioplasm, and the form of the cell has become more rounded, oval, or cubical. The nucleus is oval and is often excentrically situated; when suitably stained the coarse chromatin network, with a row of very large, deeply-stained chromatin granules, will become apparent. As a rule there is only one nucleus with a single nucleolus, and this is generally faintly stained, while the cell-body assumes a deep bluish-black colour, which makes the cell perceptible at first sight. Frequently, however, the granoplasm has partly become lost, so that the reticulum of the spongioplasm appears in one half of the cell, or more frequently on one side of the nucleus. Thus there will be an area deprived of granoplasm in the middle of the cell, and this form Marschalko has declared to be the principal type of the plasma cell. Although this view, according to Unna, may not be justified, there is no doubt that the form described by Marschalko with the unstained area in the middle is the type which will most easily be recognised. While on the whole Marschalko laid stress chiefly on the form of the plasma cells, Unna emphasised their staining property and proved that where the cells were found in heaps they were generally polygonal, oblong, or irregularly shaped, often almost ragged. In addition, the chromatin of the nucleus was always arranged along the margin in coarse grains (Fig. 7).

Unna came to the conclusion that the plasma cells originated from the local connective tissue cells, chiefly because they were never seen migrating. Marschalko was of opinion that they were

<sup>1</sup> The single granules cannot be distinguished.



emigrated, transformed lymphocytes, because all transitional forms might be found, from typical plasma cells to small mono-nucleated cells without any visible cell body, *i.e.* lymphocytes; he had also seen plasma cells in the spleen and lymph-glands of a healthy suicide and in normal animals. According to Almkvist, both authors are right, because under the name of plasma cells they deal with different kinds of cells, different even as to their origin. Later on Pappenheim, who sides with Unna in the question of origin, found in normal bone marrow large and small lymphocytes which showed the same morphological and staining properties as plasma cells. Unna, in objecting to this, says that most probably they are plasma cells and not lymphocytes. The point is that fully developed plasma cells can be distinguished without any difficulty from lymphocytes, the young ones being, on the other hand, hardly, if at all, distinguishable. According to Unna the plasma cells proliferate by amitotic division. The daughter cells are small, and exhibit a comparatively large central nucleus with a narrow, uniform, deeply-stained protoplasmic border round it. But plasma cells may also be produced by crumbling down of the "reticulum" of the spongioplasm, and the cells thus formed have a plasmic border which is irregular in outline as if it were corroded. This border is easily overlooked, and so the cells may be mistaken for lymphocytes. This form especially will be found in what is generally called "small cell infiltration" (Unna).

Further, the plasma cells easily undergo retrogressive changes which affect their appearance. Marschalko mentions that they become smaller and more oblong; the cell body becomes less susceptible of staining, while the nucleus will be deeply stained. Unna describes what he terms "*grossblasige Schaumzellen*," which have lost their granoplasm under the influence of oedema and of hyaline transformation. In general paralysis I have often been able to trace the different stages in the development of what are called fuchsin bodies ("amyloid") from plasma cells, which break down into granules that are deeply stained by fuchsin; these granules run together into big lumps, and these finally fuse into hyaline globules.<sup>1</sup>

Considering all this, we cannot expect all authors to mean

<sup>1</sup> These must not be mistaken for the globular masses which alcohol extracts from the myelin sheaths, and which give a gritty surface to preparations hardened in alcohol.

the same thing by what they call plasma cells. Thus Alzheimer found his form of plasma cells not only in the adventitial lymph-spaces, but scattered freely in the tissue, chiefly round the nerve-cells; he also described karyokinesis. Vogt adhered to Marschalko's description, and therefore excluded all forms which, staining as plasma cells, did not exhibit the forms pointed out by Marschalko. This is probably the reason why I have not so often as Vogt found lymphocytes "in great quantities" in the adventitia of the cortical vessels in paralysis, at least not in the small vessels. The typical forms of plasma cells, however, will always be found in such numbers in this disease that a few cells more or less will be of no importance. It is not even always possible to identify each single cell. But if we wish to form an opinion with regard to the occurrence of the different cell forms, the first thing required is to use appropriate methods. In Rehm's double staining (by methylene blue and alcoholic fuchsin) we have the means of distinguishing the nuclei of the plasma cells from those of the lymphocytes. In his account of the pathological anatomy of insanities, Cramer says that, by carefully studying alum-hæmatoxylin preparations, we can ascertain three kinds of nuclei in the adventitia-infiltration: (1) leucocytes, (2) endothelia of vessel- or lymph-sheaths, and (3) glial nuclei. So far as I can see, the predominant part of the infiltration consists of plasma cells, and not of the forms of nuclei just mentioned. I have not even succeeded in finding lymph-sheath endothelium in intracranial vessels. In the examination of cell forms it is of course of little use to employ methods which give to plasma cells the same appearance as glia nuclei, endothelium, and leucocytes.

The distribution of plasma cells in general paralysis is quite characteristic. They surround the vessels in large, dark masses, so that the vessel wall itself can scarcely be seen; they cluster densely, like plant-lice upon a young shoot (Fig. 3). In rapidly progressing cases especially they will be found in the dilated adventitial lymph-space of almost each vessel in the cortex, also in the subcortical white matter. During remissions they will mostly disappear, and in cases with a slow progress they will chiefly be found in the lymph-sheaths of the larger vessels. They occur over the whole cortex, in the anterior as well as the posterior portion of the cerebrum, in the central ganglia, in

the grey matter immediately subjacent to the brain ventricles, and in the cerebellum—wherever, indeed, a fresh paralytic process is going on (morbid changes of nerve-cells, degeneration of medullated fibres, glia hyperplasia). The number of plasma cells does not seem to be proportional to the degree of alteration in the meninges, and they may be found even if no alteration can be detected by the naked eye.

It has been said that the plasma cells might migrate from the vessels into the tissue. No doubt this sometimes seems to be so; I have not, however, been able to exclude the possibility that they may belong to a capillary, whose walls have not been included in the section, or which has not been stained from some cause or other. In large vessels they will never be seen moving inside the walls as long as these are healthy, and if the tissue has been injured the plasma cells will be found almost exclusively in the layers subjacent to the adventitia, having made their way into it from outside. But they will not be found close to the lumen of the vessel, nor have I seen them inside it. It must of course be admitted that possibly they do not assume their typical appearance till they come into the lymph-space, and therefore cannot be distinguished from lymphocytes in their earlier stages, when they circulate in the blood. But no elements even resembling lymphocytes will be seen passing through the vessel wall. On the other hand, if Unna is right in saying that the plasma cells originate from the local tissue cells (that is, from adventitia), then it is not quite correct to apply to them the term exudation.

Along with plasma cells, lymphocytes<sup>1</sup> will be found in the infiltrations (not often in large numbers according to my experience), as well as mono-nucleated leucocytes. Polynuclear leucocytes occur quite sporadically, in cases where there is no special reason for their appearance, such as septic infection. Some of Ehrlich's so called "fattened cells" ("*Mastzellen*") may also be found. These cells may be recognised by their markedly granular cell body; as the nucleus cannot generally be seen, the whole cell seems to consist of an accumulation of granules, and almost

<sup>1</sup> By "lymphocytes" is here always meant cell forms with one single round nucleus, which assumes a somewhat dark tint in Nissl preparations, and whose protoplasm will be visible merely as grains round the margin; it will never, at least, encircle the nucleus entirely. The leucocytes are larger, more slightly coloured, and mono- or polynuclear; the latter may have different forms.

resembles an aggregation of micro-organisms. The protoplasm granules are of different size and somewhat irregularly scattered, but each single granule may always be distinguished (which is not the case in plasma cells); these granules have great affinity for basic aniline dyes, and are generally stained metachromatically (*i.e.* they take a shade different from that of the staining fluid which has been used). In staining with Unna's polychrome methylene blue (which may be used in the same way as for plasma cells) they assume a reddish shade; when Nissl's method is used they turn a bluish-black colour. Hæmatoxylin is not suitable for their demonstration, and it is better not to embed the preparation before staining it. The form of the cells may vary; they are circular as well as flat, angular, and spindle-shaped. They may be as large as white corpuscles, or larger. The nucleus is of medium size, oval in shape; generally it is not stained, but it may take the colour of the staining fluid. It has not been definitely ascertained whether the "mast-cells" originate from the local connective tissue cells or from the leucocytes. Normally they are found in bone marrow, and are said to increase with age; they also occur in inflammations and in tumours. As far as we know at present they are of small importance in the diagnosis of general paralysis.

The nuclei of the adventitia may have increased without any concurrence of other elements. The hyperplastic nuclei often exhibit strange forms. The cell body contains considerable pigment in grains or flakes.

The other alterations of the small cortical vessels are not so constantly found. Sometimes the endothelial nuclei have distinctly increased, and now and then they seem to fill up the whole lumen; the cell body is very frequently more or less visible in the Nissl preparations. The vessel wall may have undergone a hyaline degeneration; but this term is still so vague that almost every different author applies it in his own sense. Thus capillaries are said to have undergone "hyaline degeneration" when their walls are thickened and, as it were, swollen; and the same expression is used about the smallest arteries when the connective tissue has increased and the structure has become indistinct. More rarely real deposits of hyaline matter (taking a bright red stain from eosin) will be found in the vessel wall, either in the place of the muscular coat, or more frequently

between the adventitia and media the remnants of the nuclei of which may still be faintly seen in the hyaline substance. Some vessels have contracted and seem impervious. Finally, we will find a number of newly-formed capillaries. According to experiments made in the laboratory of the Psychiatric Clinique in Heidelberg, the glia cells come forward to give support to the newly formed capillaries, their cell bodies being permeated by endothelial cells, which circumscribe the lumen of the new capillary. The protoplasmic processes from a neuroglial cell to a vessel do not become channelled, as Mendel imagined. Most of the new buds from the capillaries, however, make their way, I think, *between* the glia cells, not *through* them.

*The large intracranial arteries.*

The aggregation of plasma cells in the lymph space of the adventitia may be traced even to the large intracranial arteries. There will frequently be other alterations here, however, which are more prominent or which will be found alone. Arterio-sclerosis must of course be expected in all aged paralytics; syphilis, also, has long been thought to favour its appearance at an earlier age. But we cannot therefore say—as some authors have done—that general paralysis is the result of combined syphilis and arterio-sclerosis. Upon this occasion the obviously arterio-sclerotic alterations are less interesting than the occurrence of Heubner's syphilitic arterial disease. Anglade has never found this in general paralysis. Mendel has not seen more than 2 cases among 60, and during 14 years Cramer has not been aware of more than 3-4 well-marked specimens. But it is not quite clear whether all cases have been examined microscopically. On the other hand, Straub found this arterial disease in most of the paralytics whom he examined, and I have also been surprised to see how frequently it will be found, when carefully looked for.

Of course this difference of opinion mainly depends upon what is considered as syphilitic arterial disease. The question, therefore, is, whether we really have definite signs which enable us to distinguish between this disease and arterio-sclerotic alterations. The other "endarteritic" processes in tuberculosis, in chronic ulceration, and after ligature, etc., cannot here be taken into consideration.

Even Heubner himself was not of opinion that the arterial disease which he described was specific in regard to its anatomy, but he maintained that the process was connected with syphilis, and was anatomically different from arterio-sclerosis. He pointed out that the newly-formed layer had a larger number of cells, and was irregularly arranged in syphilis, and that there were no degenerative alterations, especially no deposit of fatty matter or lime salts. Further, the atheroma generally implicated only a part of the circumference of the vessel, but extended over a considerable portion of its length, and would mostly be found in the larger arteries, while the syphilitic arterial disease attacked in preference the small and middle-sized arteries. Generally the muscular coat was not affected by the syphilitic process, except in the extreme stages, and the infiltration of adventitia round the nutrient vessels was an accidental occurrence. But according to Heubner the muscular coat was not much altered, even by arterio-sclerosis, and if so, the form assumed was usually that of fatty degeneration. The adventitia was often infiltrated by "round cells." In arterio-sclerosis the elastic membrane became laminated, exhibiting nuclei between the laminae, and eventually it would split into 4-8 shiny bands. Also, according to Jores (p. 49), the division of the elastic membrane is *always* part of the arterio-sclerotic process, and besides this, connective tissue will be abundantly found between the laminae. He says that this splitting will only be found in syphilitic endarteritis when the endarteritis has developed in slightly arterio-sclerotic vessels.

Leaving out of the question the vessels which are evidently arterio-sclerotic (*i.e.* vessels charged with fatty matter and lime salts), the diseased cerebral arteries of large calibre (art. basilaris, fossæ Sylvii, etc., and their first branches) in general paralysis present, as far as my experience goes, the following aspect (Fig. 5). The endothelium does not seem to be altered; the nuclei form as usual only one layer. Under the endothelium may frequently be seen a structure which in its form resembles an elastic membrane, but which does not give the staining reaction characteristic of elastin, nor will those remnants of nuclei be found which in all text-books are called "fenestra." But a little further out in the vessel wall a typical elastic membrane will be found, which evidently is the original

membrane of the vessel. Between this and the endothelia is a *newly-formed layer*, consisting of a fibrillar ground substance and numerous cells. The intercellular substance is, as a rule, poorly developed, but it may consist of huge bundles of fibres; these will not generally give the reaction of elastic tissue, but sometimes they may do so. In preparations stained with the ordinary stains for nuclei, the nuclei of the newly-formed layer sometimes resemble endothelial nuclei, sometimes those of muscle cells, but generally they are unlike both. The *elastic membrane*, which will be found in its place close to the muscular coat, may show its original appearance, but frequently the folds are not so regular as formerly; the membrane may in part be quite straight under the newly-formed layer, and in some places it has taken no staining, so that its circular line looks broken. In the place where it seems to be least prominent, elastic fibres have developed, generally on the inside of the membrane, but sometimes on the side facing the muscular coat. In the elastic membrane two layers may be distinguished as usual; the inner layer will be stained a deep red by Weigert's resorcin fuchsin, and the outer layer black by Heidenhain's iron hæmatoxylin. In an early stage of the arterial disease the inner layer is thicker, as if swollen and about to be split. Between the divided layers nuclei will appear. It seems as if this splitting of the membrane, with the appearance of nuclei between the layers, is the alteration with which the whole process begins, and this might explain why the condition in the brain vessels just described will be seen in the arteries only and not in the veins, which, as a rule, have no elastic membrane. Heubner thought the newly-formed elastic membrane was derived from the endothelium. In my opinion, it has its origin in the already existing membrane. It need not, therefore, be simply a splitting of this membrane, since the laminæ, taken together, may be thicker than the original membrane. Those sheets or elastic fibres which split off are liable to retrograde changes, and then they will no longer give the reaction of elastic tissue. The wavy fibres of the layer may partly, however, be common collagenous tissue.

The new layer which develops inside the inner lamina of the elastic membrane projects forward into the lumen, and may become prominent at a circumscribed spot of the wall, but will generally follow the whole circumference of the vessel. In

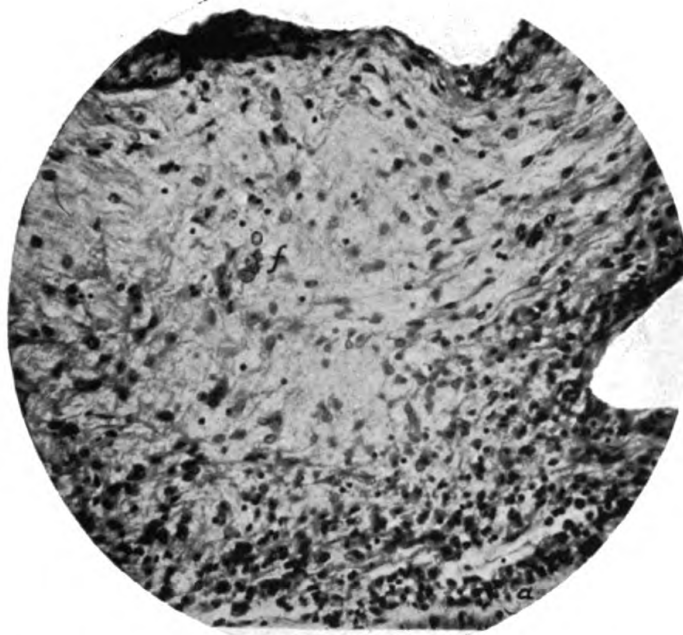


FIG. 1.—Considerable thickening of pia arachnoid, with proliferation of nuclei and connective tissue fibres. *f*, a collection of fibroblasts. *a*, the wall of an artery; in adventitia with immediate surroundings numerous plasma cells. Hæmatoxylin-eosin. Leitz lens 6, oc. 1.

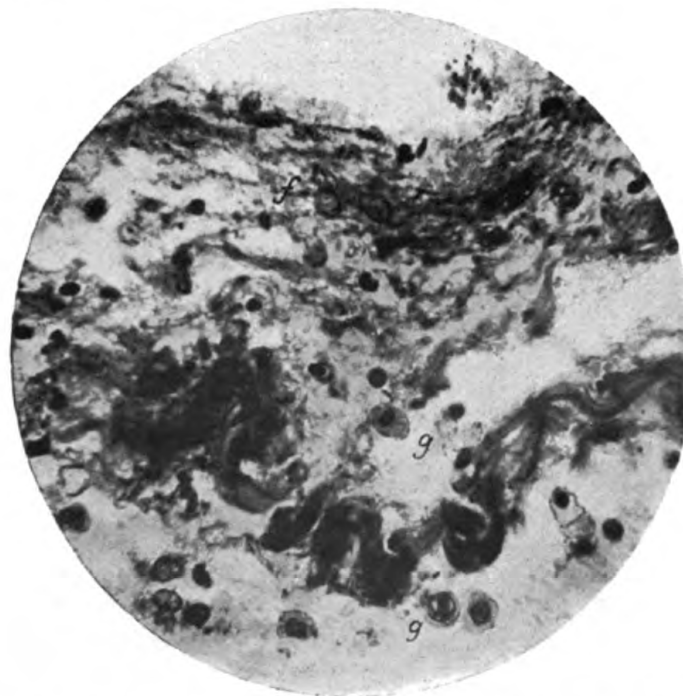


FIG. 2.—Pia arachnoid. In the lower part a bundle of elastic fibres, probably belonging to a vessel not in section. On both sides "Gitterzellen" (*g*); some other cells are certainly plasma cells. *f*, nuclei of fibroblasts. Hæmatoxylin, and then the resorcin-fuchsin method of Weigert. Oil immersion  $\frac{1}{2}$ , oc. 1.





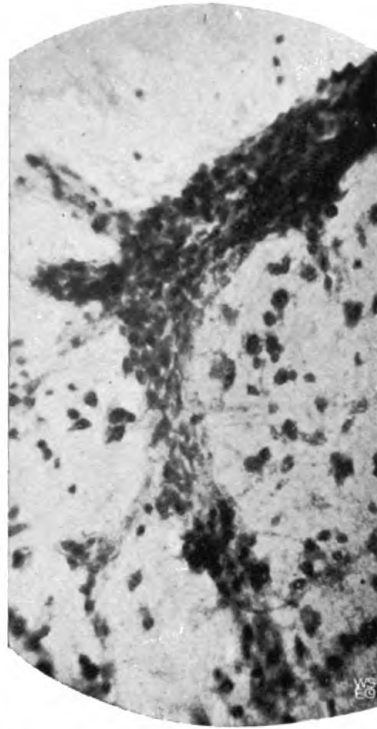


FIG. 3.—Plasma cell-infiltration of a vessel in the cerebral cortex. In the vicinity spider cells, sending protoplasmic prolongations to the vessel wall. Bevan Lewis aniline blue black. Lens 6, oc. 1.

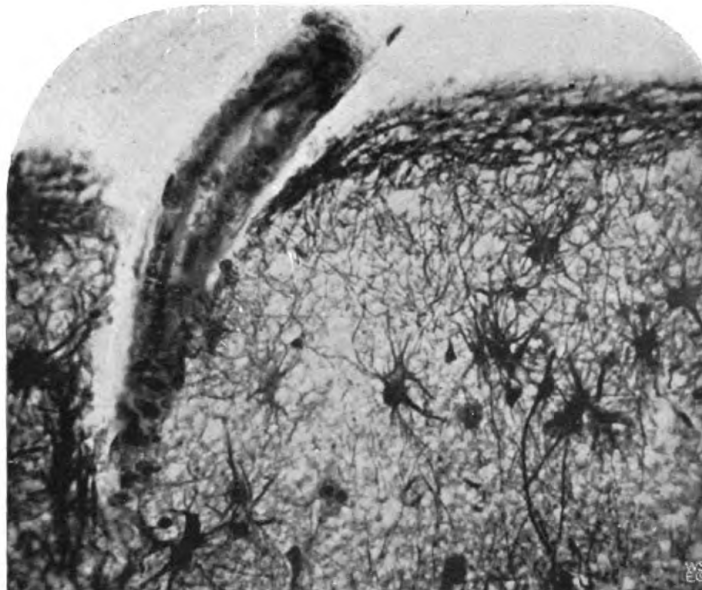


FIG. 4.—Spider cells in the outermost layer of the cortex, with dense network of glia fibres. From a case of juvenile paralysis. The artery shows no change. Method of Held. Lens 7, oc. 1.



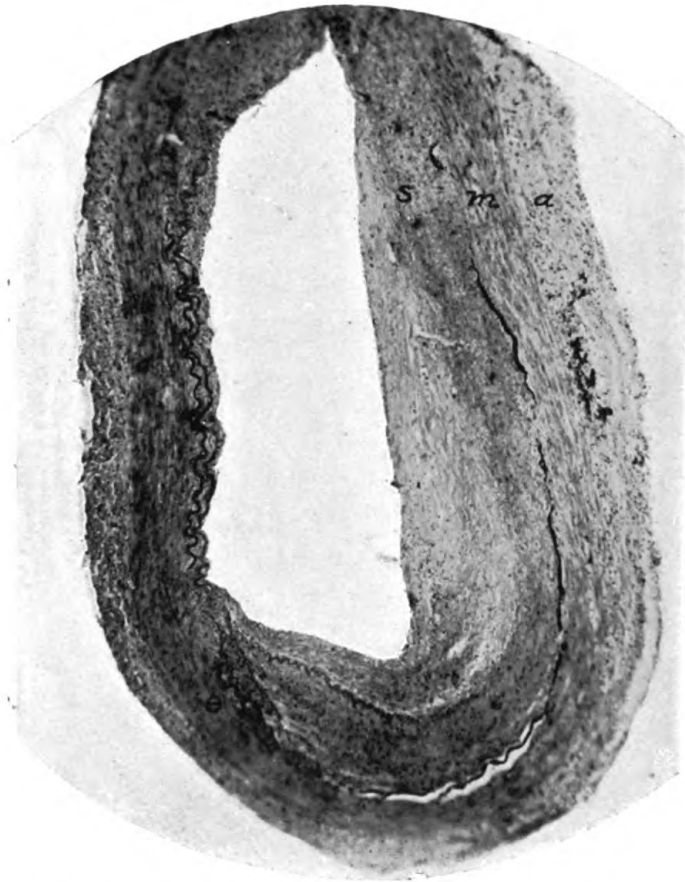


FIG. 5.—A probably syphilitic cerebral artery; *s*, newly formed sub-endothelial layer; *m*, muscular coat, considerably narrowed. The elastic membrane (*e*) partly interrupted, nearly straightened in places where the newly formed layer is most developed. On the opposite side of the artery there are several laminae split off from the membrane towards the lumen. Heidenhain's iron-haematoxylin. Lens 3, oc. 1.



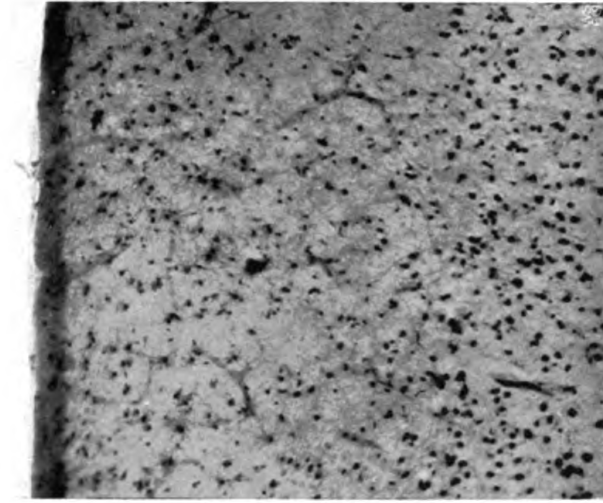


FIG. 8.

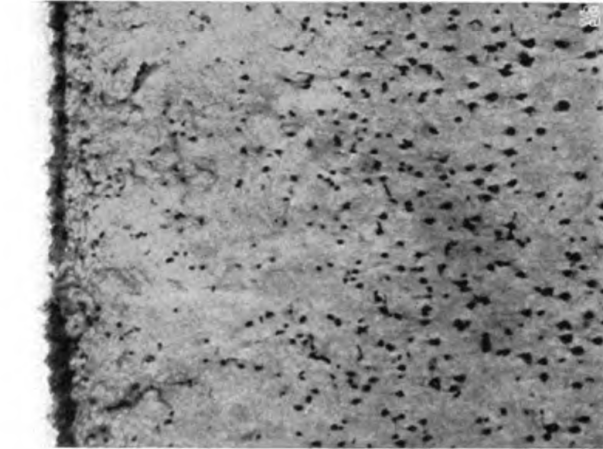


FIG. 7.

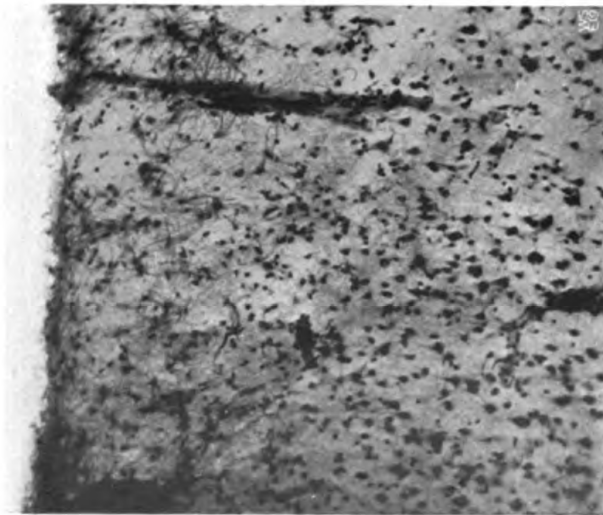


FIG. 6.

FIGS. 6-8.—Subpial felting and upper layers of the cerebral cortex in cases of paralytic (fig. 6), senile (fig. 7), and epileptic (fig. 8) dementia. Fig. 6 shows shrinkage of the cortex, infiltration of the vessel sheaths, and numerous large spider cells. Such cells are fewer and smaller in figs. 7 and 8. Bevan Lewis method. Lens 3, oc. 4.





FIG. 9.—Plasma cells (Marschalko's type) round a capillary of the cerebral cortex. Beneath the lowest plasma cell an endothelial nucleus. Nissl's method. Oil immersion  $\frac{1}{2}$ , oc. 4 (1000 $\times$ ).



FIG. 10.—Staff cells. One of these is reproduced from a rather dark copy in order to make the branching cell body more distinct. Nissl's method. Oil immersion  $\frac{1}{2}$ , oc. 4.





general paralysis considerable narrowing or entire obliteration of the lumen will seldom occur. The layers grow at intervals and probably very slowly. Several (2-3) layers of different ages may frequently be distinguished, each of which will be limited by an undulating line, that marks an elastic lamina which has been separated at some time or other. On the newly formed layer thrombosis may take place. Later on the obliterating masses become vascularised; 3-4 or more new large lumina, each having a separate muscular coat, may be seen. There are no traces of fatty or calcareous degeneration.

The *muscular coat* has not been altered where the subendothelial layer is only very slightly developed, but it will generally become narrower in the place corresponding to the newly-formed layer, and in a later stage of the arterial disease the muscular coat will sometimes become quite wasted.

The *adventitia* is now and then uniformly thickened, almost exclusively by increase of the connective tissue fibres, which may form huge bundles; the elastic fibres have not often increased. But there need not be any alteration here, even if the newly-formed layer under the endothelium is somewhat thick. Frequently, however, the adventitia has been infiltrated by alien cells, especially in the outer layer, and not unfrequently they have penetrated to the adjacent parts of the wall in those vessels where the muscular coat is atrophic, and where abundant elastic tissue has been developed in the subendothelial layer. In all likelihood the cells are lymphocytes and plasma cells. The infiltration of the adventitia is probably more directly connected with the paralytic process, while the syphilitic vascular lesion (Heubner) does not in itself relate to general paralysis.

In general paralysis the syphilitic arterial disease will be found not only in the intracranial arteries, but also in other parts of the arterial system. In syphilitic individuals without any trace of mental disease it has been found even a few months after the infection. The anatomical picture of the arterial disease seems mostly to indicate a slow and irregular progress with partial repair, and this fully agrees with the fact that in the greater number of my cases the lesion did not seem to have caused any evident disturbance in the function of the vessel.

The reasons for my opinion that the arterial disease just described is caused by syphilis and not by arterio-sclerosis are:

first, that the changes resemble those found in the intracranial vessels in a case of syphilitic brain disease occurring at about seventy years of age, where no gross alterations of the aorta were to be seen; secondly, the absence of fatty or calcareous deposits in the newly-formed layer, even where this was markedly developed; and, finally, because the original elastic membrane was always recognisable, and was never completely divided into equally delicate laminæ. Even in arterio-sclerosis, however, this mode of splitting may not always be found. In general paralysis the plasma cells will, of course, afford no aid to the differential diagnosis between the two vascular diseases, as the vessel may be arterio-sclerotic before the paralysis begins; but in arterio-sclerosis *alone* the plasma cells will never be found in large numbers.

Nothing definite can be said about the nature of the vascular changes till further investigations have been made, and we must be prepared to find that most heterogeneous processes are included in what we now class under the common name of arterio-sclerosis.

(*To be continued.*)

---

## Abstracts

### ANATOMY.

**A CONTRIBUTION TO OUR KNOWLEDGE OF THE STRUCTURE (298) OF THE NERVE CELL.** (Contributo alla conoscenza della struttura delle cellule nervose.) GEMELLI, *Riv. Speriment. di Freniatria*, Vol. xxxii., Fasc. 1-11, p. 212.

IN this paper, which is a preliminary communication, the author reviews the work already done upon the neurofibrils and proposes to answer three questions: (1) If long fibrils exist; (2) the existence and the form of the endocellular reticulum; (3) the combination of the long fibrils to form an endocellular network or their independence.

The research when finished will embrace vertebrates as well as the lower forms of life, and the author recognises the importance of using one method which will give constant positive results in all. With this object he has employed a modification of the

osmio-bichromate-silver nitrate method, and gives the result of his observations on worms.

The fibrils which enter the cell from the nerve process are very fine, and divide into two or three rami, which, uniting with others, help to form a meshwork. This completely surrounds the nucleus. In the worm there are no long fibres, all fibrils ending in anastomosis.

DAVID ORR.

**CADAVERIC ALTERATIONS OF THE NERVE CELLS STUDIED  
(299) BY THE METHOD OF DONAGGIO. (Le alterazioni cada-  
veriche delle cellule nervose studiate col metodo di Donaggio.)  
V. SCARPINI, *Riv. Sper. di Fren.*, Vol. xxxi., Fasc. 3-4.**

IN this investigation the author examined pieces of the spinal cords of rabbits, which had been removed from the spinal column immediately after death, and kept in a damp room at a temperature of 15° C.

Fragments about 5 mm. thick were taken every two hours and placed in fixing fluids. The results showed that no changes of any importance had taken place in tissues which had been exposed for twenty-four hours. After a little longer exposure the fibrils, peripheral and central, had become less distinct and had broken up into very fine granules. At the same time the nucleus and the nucleolus had become stained, the cell had lost its processes, and had gradually become disintegrated.

These changes differ from purely pathological alterations, in that the contour of the cells had altered; they were uniform throughout the cell, and when the granular condition was complete the nucleus was also stained. On the other hand, in pathological conditions the contour of the cell is preserved, the reticular fibrils break down before the long peripheral fibrils, and the nucleus never becomes stained.

R. G. ROWS.

**ON SOME PRIMARY ALTERATIONS OF THE ENDOCELLULAR  
(300) FIBRILLARY RETICULUM AND OF THE LONG FIBRILS  
IN THE CELLS OF THE SPINAL CORD. (Su alcune alte-  
razioni primitive del reticolo fibrillare endocellulare e delle  
fibrille lunghe nelle cellule del midollo spinale.) V. SCAR-  
PINI, *Riv. Sper. di Fren.*, Vol. xxxi., Fasc. 3-4.**

THE author first examined the nerve cells of animals which had been killed by the inhalation of ethyl chloride. The results, as far as the fibrillæ were concerned, were negative.

He then described the condition of the cells of the cord after

compression of the aorta. These results varied, of course, with the duration of the anæmia produced. With an anæmia lasting for twelve minutes, even if repeated three times, there was very little departure from the normal. If, however, the anæmia lasted a little longer than that, the network of fibrils became less distinct, the fibrils themselves less regular, and some fragmentation could be seen; vacuolation of the cell also occurred. The fibrils in the centre of the cell were more affected than those at the periphery.

After an anæmia of three hours, the reticulum had lost its structure, and the fibrils throughout the cell had undergone a granular disintegration. With still longer compression of the aorta, softening of the cord was produced.

Other methods have shown that the chromophile elements of the nerve cells react to morbid agents more readily than do the fibrils. If, however, the lesion of the chromophile elements is not accompanied by much injury to the fibrils, recovery of the cell is possible, although the function of the cell may have been temporarily almost lost; but if the fibrils have been damaged to any extent, *i.e.* if they have undergone this granular change, recovery is impossible.

R. G. ROWS.

### PHYSIOLOGY.

#### CONTRIBUTIONS TO THE PHYSIOLOGY OF THE JAW-MOVE-

(301) MENTS. (*Beiträge zur Physiologie der Kieferbewegungen*)

RIEGNER, *Archiv. f. Anat. und Phys.*, 1906, p. 109.

THIS paper deals with the action of the individual jaw muscles of *Macacus Rhesus* in supplement of an earlier research by the same author on the effect produced by drawing upon the jaw muscles of the human body, carefully isolated after death. The method employed was to kill the ape with chloroform, and then to stimulate electrically the muscles, which retained their full contractility for an hour after death. The action of each of the seven muscles is shortly given, and found to be in general accord with the results obtained on the human subject.

JOHN D. COMRIE.

### PATHOLOGY.

#### CONCERNING DEGENERATION AND REGENERATION OF

(302) PERIPHERAL NERVE-FIBRES. (*Sopra la degenerazione e rigenerazione [in seguito al taglio] delle fibre nervose periferiche.*)

CARLO BESTA, *Rivista Sperimentale di Freniatria*, Vol. xxxii, p. 99.

FROM experimental researches, consisting in the division of peripheral nerves, turning up the central segment and suturing it to a

muscle, thereby preventing the reunion of the central with the peripheral end, the author comes to the following conclusions as regards the process of degeneration:—All the fibres in the peripheral segment undergo degeneration. The process commences in the axis-cylinder, firstly as a swelling, then as a granular degeneration of the fibrils composing the axon. Then follows a destruction of the supporting stroma of the axis-cylinder, and, parallel with this, a proliferation of the cells of the neurilemma. In the place of each fibre there comes to be formed a protoplasmic thread, rich in nuclei, which after a time becomes attenuated and is at last reduced to a mere chain of delicate bipolar elements. The process is identical in young animals and in adults. The writer has never seen any migration of leucocytes into the nerve-fibre during the degenerative process, and believes the phagocytosis to be carried out entirely by the proliferated neurilemma cells.

In the process of regeneration, the author describes how the neurilemma cells proliferate within the old fibres and form themselves into bundles of protoplasmic threads running longitudinally. The lower end of the axis-cylinder of the proximal segment then becomes attached to one of the newly-formed protoplasmic threads, and thereby exercising an influence on it, a delicate new axis-cylinder is laid down in the protoplasmic thread. In this way the new fibres, though not branches or prolongations of the central axis-cylinder, are formed under its immediate influence. Besta's results therefore support the pluri-cellular origin of regenerated fibres, whilst he maintains that the central segment exercises a stimulating influence on the process.

PURVES STEWART.

**ON THE FUNCTIONING OF DEGENERATE MUSCLES.** Third (303) Paper. (Mechanical Work and Potency.) (*Sulla Funzione dei Muscoli Degenerati. IIIa Comunicazione. [Lavoro meccanico e potenza.]*) Dr GUIDO GUERRINI, *Lo Sperimentale*, May-June 1906.

IN this paper Dr Guerrini continues his examination of the functioning of muscles in a state of fatty degeneration.

Two points have to be considered: (1) The mechanical work done by the muscle; and (2) The mode in which a muscle raises a weight to a certain height and there sustains it.

A short account of the work already done by others with reference to the first point is followed by a description of the machine which the author himself made use of.

The experiments were made on edible frogs, the gastrocnemius being the muscle used. The weight employed was one gramme;

it was found that heavier weights gave almost exactly the same total amount of work, though naturally the individual elevations were less. The number of stimuli was two per minute; slackening this rate did not affect the tracing, while increasing it was apt to cause the degenerate muscles to enter the state of contracture. Moreover, the author desired not to have phenomena due to fatigue superposed upon the tracing, his object being to bring about the material exhaustion of the muscle uncomplicated by the specific action of those products of muscular catabolism which play so great a part in fatigue.

Records of twenty experiments follow. They show that the degenerate muscles, as compared with the normal, give a tracing in which the ordinates are both shorter and fewer in number. Moreover, there is in the former case a greater irregularity in the amplitude of the movements, and the initial rise of the curve is either wanting or of much shorter duration.

From these facts the following conclusions are reached: (1) That whilst in normal muscles (sufficient interval being allowed) to identical stimuli correspond contractions equal in amplitude and form, in degenerate muscles contractions corresponding to identical stimuli differ in amplitude and form. (2) Whilst in normal muscles identical stimuli repeated at short intervals bring about increased excitability, this does not occur in degenerate muscles. (3) Hence it appears that whilst in normal muscles the excitability persists with remarkable constancy, in degenerate muscles it oscillates between fairly wide limits.

The decrease of mechanical work in the case of a degenerate muscle is very considerable; thus while one cm. of normal muscle gives 119.97 cm.-grms. of work, one cm. of degenerate muscle gives only 74.36.

In order to study the second mechanical property of the muscle—which in a former paper Dr Guerrini called muscular potency—the area of the tetanic curve was calculated for normal and degenerate muscles respectively. The method of experiment is described, and figures referring to seventeen experiments given.

The results show that the muscular potency of a degenerate muscle is much less than that of a healthy muscle, the ratio being 23:70.

In both muscles in the second tetanic contraction the muscular potency is less than in the first, but whilst in a normal muscle it decreases to about one-half, in a degenerate muscle it decreases to about one-fifteenth.

Finally, an examination of the muscular potency represented by the sum of all the tetanograms shows that the degenerate muscle possesses barely one-tenth of the potency possessed by the normal muscle.

The paper concludes with a short enquiry into the causes of the diminution of power of work and of muscular potency shown by degenerate muscles and with a bibliography of the subject.

MARGARET DRUMMOND.

#### **HISTOPATHOLOGICAL CHANGES OF THE CEREBELLUM IN**

(304) **GENERAL PARALYSIS.** (Die histopathologischen Veränderungen des Kleinhirns der progressiven Paralyse.) E. STRÄUSSLER, *Jahrbuch für Psychiat. und Neurol.*, Band xxvii., 1906.

THIS is a long enquiry into the changes which are met with in the cerebellum in general paralysis. Microscopically the evidences of disease are less prominent than in the cerebrum. The meninges are not thickened to such an extent, nor do the vessels show so much change. Microscopically the changes in the meninges consist of a collection of cells around the vessels in the outer layers, while in the inner layers there is an infiltration of the tissues.

The alterations in the vessels the author considers to be secondary.

There is a considerable discussion with regard to the relation of syphilis to the changes in the vessels, and after stating that the proportion of cases in which a syphilitic arteritis has been found varies from 82% in the cases of Straub to 47% in those of Chiari, and 13.5% in his own cases, he expresses the opinion that syphilitic alterations in the vessels cannot be accepted as the cause of the lesions found in the nervous tissues.

The lesions of the nervous tissues consist of a loss of nerve cells and fibres, which is followed by an overgrowth of the neuroglia, and a shrinking of the convolutions. Vacuolisation of the nerve cells is common. The regions of the cerebellum which are most affected are situated around the semi-lunar sulcus, the lobus centralis and the lobus superior anterior, and parts of the inferior worm, the nodulus and uvula. These lesions are at first limited to the superficial portions, and only in the advanced stages of the disease do they attack the deeper convolutions.

With regard to their origin, the author suggests that they are primary degenerations produced by toxins circulating in the cerebro-spinal fluid, and, as a matter of fact, it is precisely the superficial portions, which are most exposed to the cerebro-spinal fluid, which are most affected. The other changes met with must be considered as secondary.

Lastly, the author expresses the opinion that, although there is no close relationship between the intensity of these cerebellar



changes and the motor symptoms of general paralysis, they do sometimes play an important part in the determination of the clinical picture.

R. G. ROWS.

# **EXTIRPATION OF THE LOWER HALF OF THE SPINAL CORD**

(305) **AND ITS RESULTS.** (Ueber die Extirpation der unteren Hälfte des Rückenmarks und deren Folgeerscheinungen.) L. R. MÜLLER, *Deut. Zeitschr. f. Nervenheilk.*, Bd. 30, H. 5-6, S. 413.

A DOG had its whole lower cord, except the conus terminalis, removed in three stages up to the level of D 9. After the primary disturbances had passed off there was automatic emptying of the bladder and rectum.

Erection and seminal ejaculation were possible with the upper lumbar cord intact, but not when it was removed.

The dog was killed after two years. Over the hinder part of the body the skin and hair were then as healthy as over the normal anterior part. Some pressure sores which had formed early over the feet had healed. The subcutaneous fat was also as abundant as elsewhere.

The muscles of the hind limbs were almost entirely fatty; a little fibrillary substance could still be seen microscopically. The muscle spindles were for the great part intact. The bones of the hind limbs were more rarified than those of the fore. In the nerves, nearly a third of the fibres still had medullary sheaths, although these were somewhat granular. Probably they were fibres from the cells of the posterior root ganglia, which were undistinguishable from normal ganglia cells.

In the conus there were no marked signs of secondary degeneration except in the dorso-medial tract. This tract was also affected in the upper dorsal cord, but no degeneration could be traced into the cervical region.

J. H. HARVEY PIRIE.

# **A SERUM REACTION OCCURRING IN PERSONS SUFFERING**

(306) **FROM INFECTIVE CONDITIONS.** LEWIS C. BRUCE, *Journ. Ment. Sc.*, July 1906.

THE author, having noticed in the course of making observations upon the opsonic indices of the insane that the serum in many of the cases agglutinated the red blood corpuscles of a healthy person, examined the serum of some recent cases of insanity, and found that the serum of all persons suffering from mania with confusion, the mania or depression of folie circulaire, katatonia,

hebephrenia, and the excitement associated with epilepsy, invariably gave this agglutinative reaction. Whereas cases of insanity due to other causes than bacterial infection, such as melancholia of metabolic origin, systematised delusional insanity, etc., did not give this reaction.

The writer also observed that the red blood corpuscles of a patient in whom this agglutinative reaction is present are protected against the action, not only of the agglutinine in the patient's own blood, but also against the agglutinine in the serum of another patient.

The substance which causes this agglutinative reaction is thermostable, and it is not the same substance as the agglutinine. It also gradually disappears from the serum in some cases—the reaction may be quite definite in a serum recently obtained from a patient, while the same serum six hours later will give a very indifferent reaction.

By means of this test it is possible to divide cases of insanity into two great groups, infective and non-infective.

H. DE M. ALEXANDER.

### CLINICAL NEUROLOGY.

**TWO BROTHERS AFFECTED WITH PRIMARY PROGRESSIVE (307) MYOPATHY. ADDITIONAL NOTE.** (*Deux frères atteints de myopathie primitive progressive. Note additionnelle.*)  
NOICA, *Nouv. Icon. de la Salpêtr.*, March-April 1906, p. 151.

THIS article deals with the sensory disturbances present in two cases of myopathy which had been previously reported. The first case showed tactile anæsthesia over the whole surface of the body, with the exception of the head, neck, palms, and soles. There was also hypoalgesia and delayed perception of pain over practically the whole of the same extensive area. Sensibility to heat was diminished, but sensibility to cold was unaffected. The stereognostic sense, and sight, hearing, taste, and smell were normal. In the second case the sensory symptoms were less marked but similar in nature, and although at first the skin of the limbs was alone affected, the distribution eventually extended until it was almost identical with that noted in the first patient. Examination of a portion of a cutaneous branch of the anterior tibial nerve removed during life from one of the patients revealed very definite pathological changes. The author discusses the relation of the sensory phenomena and nerve changes to the muscular atrophy, and although unable to arrive at a definite conclusion, he thinks

it probable that the nerve lesion has been superadded to the muscular one, and that no etiological connection between the two exists.

HENRY J. DUNBAR.

**ON A CASE OF "CRUTCH PARALYSIS."** (*Sur un cas de "paralyse des béquilles."*) F. SOCA, *Nouv. Icon. de la Salpêtr.*, March April 1906, p. 171.

THIS article is based on the case of a soldier, aged 30 years, who, while recovering from a wound of the right leg, was obliged to employ crutches to enable him to walk. After a few days of their use he began to experience tingling and numbness, succeeded by progressive loss of power in the whole of the right arm. A fortnight after the commencement of symptoms, the arm was found to present the typical signs of musculo-spiral paralysis. On careful consideration, however, it was discovered that there was indisputable evidence of affection of the whole brachial plexus, as shown by paresis of the muscles supplied by the median and ulnar nerves, and also of the biceps, brachialis anticus, coraco-brachialis, deltoid, subscapular, pectoralis major and minor, supraspinatus, infraspinatus, rhomboids, levator anguli scapulæ, and serratus magnus. There was also anæsthesia to pain, heat, and cold, with unaffected tactile sensibility over the whole arm. The accepted theory of the mechanism of crutch paralysis is that the musculo-spiral nerve is compressed against the humerus by the direct action of the crutch. The distribution of the paralysis in this case shows the lesion to be one affecting the nerve roots—not of the peripheral nerves nor of the brachial plexus. The cause of this root lesion cannot be direct compression, and is almost certainly traction, the end of the crutch acting as a pulley. The author has demonstrated this mechanism by experiment on the cadaver. The fact that the musculo-spiral nerve is most affected is probably explained by the direct pressure of the crutch, a pressure to which the other branches of the plexus are less exposed.

HENRY J. DUNBAR.

**ALCOHOLIC NEURITIS.** CORIAT (of Boston), *Am. Journal of* (309) *Insanity*, April 1906.

THE clinical histories of seventeen cases are given, and some of the most recent literature on the subject is reviewed at length. The conclusions concerning the mental disturbances associated with alcoholic neuritis are as follows:—

With eye muscle palsies, which may be of either central or peripheral origin, there is often an implication of the higher central

neurones, and a consequent delirium, with marked allopsychic disorientation, or the development of a fabricating psychosis. Again, peripheral neuritis may be associated with a posterior column degeneration, and bear a close resemblance clinically to tabes, or the central involvement may exist by itself, and give rise to a characteristic terminal disorder. Ordinary peripheral neuritis is, as a rule, associated with some central disorder. Cole believes that the central disorders consist in an axonal reaction of the Betz cells, with a degeneration of their connecting pyramidal tracts, and a posterior column degeneration similar to that seen in tabes.

Korsakow's psychosis may occur without any signs of peripheral neuritis, and be caused by other factors than alcohol. A delirious state of very acute onset, strongly resembling delirium tremens, may occur, and when associated with peripheral neuritis, may show marked disorientation, poor retention, defective memory for recent events, and confabulation. The course of the disease is usually acute, and may end with the motor disorders of a terminal central neuritis. There is still another type of Korsakow's psychosis of acute onset, with isolated neuritic symptoms, which progresses rapidly to recovery. In true delirium tremens which shades into a fabricating psychosis, recovery is usually not complete, some mental reduction follows, or the delirium may subside rapidly and leave a slowly improving neuritis. If neuritic symptoms appear during delirium, suggestibility and marked fabrication are always superimposed.

Another group of cases show a protracted course, and ultimately exhibit all the clinical symptoms of a central neuritis, *i.e.* emaciation with diarrhoea, rigidity, and twitchings.

In the pure acute hallucinosis with neuritis from the start, or developing later in the course of the disease, the outlook for recovery is very favourable.

Depressive delirious states of rapid course with or without polyneuritis or a dreamy hallucinatory confusion may exist, always without fabrications or amnesia. Finally, there are cases resembling at first an alcoholic deterioration process, exhibiting recent memory defect, and running a slow course, which show an almost complete recovery parallel with the disappearance of physical signs.

C. H. HOLMES.

**TABES AND AORTIC ANEURYSM.** (*Tabes et anévrysme aortique.*)  
(310) DEBOVE, *Journal des Practitiens*, June 9, 1906.

THIS is the account of a case which presented signs indicative of tabes or of aortic aneurysm. Briefly these were loss of weight, and gastric crises, lasting over eight years, loss of muscular power,

hiccough, abolition of tendon reflexes, slow pupillary reaction to light, great muscular hypotonus, severe pain down the right arm, but no shooting pains, no ataxia, no Rombergism.

The writer in passing remarks upon the denial by Babinski, Vagnez, and others, that pupillary dilatation in cases of aneurysm is due to compression within the thorax, these writers attributing it to syphilitic eye changes. Being in a dilemma as to the diagnosis, he draws attention to the importance of two recent diagnostic methods, viz. lumbar puncture and skiagraphy. The former discovered many lymphocytes in the arachnoid fluid, enabling a diagnosis of tabes to be made, while the latter showed with certainty a large aneurysmal dilatation of the aorta.

JOHN D. COMBIE.

**A STUDY OF PARAPLEGIAS FROM RETRACTION IN OLD (311) PEOPLE.** (*Étude sur les paraplégies par rétraction chez les vieillards.*) P. LEJONNE and J. LHERMITTE, *Nouv. Icon. de la Salpêtr.*, May-June 1906, p. 256.

PARAPLEGIA from muscular and tendinous retraction due to chronic myositis in old people has, on account of its close resemblance clinically to paraplegias of other origin, not received the attention which it merits. The disease has been to a great extent confused with chronic rheumatism and with the contractures following prolonged want of use in paraplegia. It is essentially a disease of old age, the majority of the cases being over 75 years, and women are more frequently affected than men. Although not a necessary etiological factor, confinement in bed from fracture or other injury, or from weakness, physical and mental, is an important predisposing cause. The onset of the disease is insidious, and is usually preceded by vague pains and cramps in the legs. After a few months the patient is unable to walk on account of weakness of the legs, and at this stage the muscles are found to be soft, flabby, giving a brisk reaction to mechanical stimulation, but not diminished in volume. Atrophy and retraction follow rapidly, the legs taking up and becoming fixed in a position of flexion and adduction. The thighs are flexed on the pelvis, the legs on the thighs, and the feet are extended. The atrophied muscles feel like tense cords, and are extremely sensitive to touch, but there is no tenderness along the lines of the nerve trunks. The muscles of the trunk, neck, and arms, although atrophied, are not affected by the disease until late on in its course, and then not to the same extent as the arms. There is no qualitative change in the electrical reactions. The joints are unaffected except by changes secondary to their position and immobility. Emaciation becomes extreme, but the intellect remains clear until

just before death, which occurs in a few years from the first symptoms. The differential diagnosis is most difficult in the early stages. There are many conditions with which it may be confused, but careful examination will usually make a diagnosis possible. In particular, the electrical reactions must be thoroughly tested. The pathology of the condition is described in great detail. It consists of an atrophy of the muscle fibres accompanied by fatty and fibrous degeneration and a sclerosis and retraction of all the tissues of the muscle and tendons. No lesion is discoverable in the central nervous system or in the peripheral nerves to which the muscular condition could possibly be secondary.

HENRY J. DUNBAR.

**ON THE PATHOLOGY OF THE EPICONUS MEDULLARIS.** (Zur (312) *Pathologie des Epiconus medullaris.*) L. MINOR, *Deut. Zeitschr. f. Nervenheilk.*, Bd. 30, H. 5-6, S. 395.

THE author defines the epiconus as that part of the spinal cord, including the fifth lumbar and first and second sacral segments and the epiconus region, as these segments with their nerve roots.

The present type of epiconus lesion is met with in cases of poliomyelitis, and the next best in traumatic central hæmatomyelia. In this paper two cases of the former and one of the latter are described.

The characteristic features are: atrophic paralysis of calf muscles and peronei, particularly the latter, with steppage gait and tendency to talipes; Achilles jerk lost, knee-jerk present and increased; no affection of sphincters or sensibility.

If the lesion extends into L 4 at all, the glutei and adductors of the thigh are also affected.

J. H. HARVEY PIRIE.

**A CONTRIBUTION TO OUR KNOWLEDGE OF TRAUMATIC CONUS LESIONS.** (Ein Beitrag zur Kenntnis der traumatischen Conus-läsionen.) FISCHLER, *Deut. Zeitschr. f. Nervenheilk.*, Bd. 30, H. 5-6, S. 364.

IN this paper the author describes two new cases of pure conus lesions, and reviews some nineteen cases (nine with section) from the literature.

The usual symptoms are: a temporary paraplegia; motility usually returns early and completely, but sensory disturbances in the region of the anus, perineum, and external genitals are longer lasting and may be permanent. Dissociation phenomena frequent.

Disturbance of function in the bladder, rectum, and sexual organs may be temporary or permanent.

The majority of cases follow on falls from a little height, either on the gluteal region directly, or on the feet first and then on the back. A pure conus lesion may result without any injury to the vertebral column. The mechanism seems to be a traction through the nerve roots of the cauda equina or the conus (where the lumbar curvature is increased), with tearing of tissue and subsequent effusion of fluid. Even when there is also damage to the vertebræ this mechanism may act.

J. H. HARVEY PIRIE.

#### **A CONTRIBUTION TO THE STUDY OF AMAUROTIC FAMILY**

(314) **IDIOCY.** F. J. POYNTON, J. H. PARSONS, and GORDON HOLMES,  
*Brain*, 1906, Part 114, p. 180.

THE clinical histories of three cases are recorded. All three children belonged to Jewish families, and no other child was affected in any of the families. In each of the three cases the symptoms of the disease came on at about six months of age, and death occurred when the children were between a year and eighteen months old. The history and the course of the disease was typical in each case. The symptoms began with arrest of development and evidence of general weakness, inability to sit up without support, or to move about in a normal way, and failure of vision. Examination showed weakness and slight spasticity of the limbs, especially of the lower extremities, increase of the deep reflexes, and extensor plantar responses were obtained in at least one case. There was also marked mental deterioration, advancing as the disease progressed to complete idiocy. The characteristic retinal changes, the frequently described cherry-red spot at the fovea with a white halo around it, were present in the three cases. In two of the cases there was, in addition, optic atrophy, but the discs of the third case appeared normal.

The central nervous systems of two of the cases were obtained for examination. There was nothing noteworthy in the macroscopical appearance of either brain, except slight wasting of the gyri, especially in the frontal lobe, and an unnatural firm consistence of the whole forebrain to touch. The following methods were employed in the microscopical examination: Nissl's and Bielschowsky's for investigation of the cells, Weigert's and Marchi's for the medullated fibres, and various portions of the central nervous system of one case were, in addition, stained by Weigert's neuroglia method.

The changes found were practically identical in the two cases. Not a single unaffected cell remained in any part of the central

system, in the dorsal root ganglia, or in the retina. The nerve cells appeared swollen and inflated, their nuclei, which were often shrunken, generally lay excentric, and they had, as a rule, almost disappeared, so that the only part of the cytoplasm which stained by the basic aniline dyes was a finely granular substance around the nucleus. The nature of this substance is doubtful; it resembled a degeneration product, but stained deeply with hæmatoxylin, and was not coloured by osmic acid or any of the fat stains. Many of the larger cells were, in addition, vacuolated. The cells of the cerebellum were, on the whole, less affected than those of any other part of the central nervous system. The neurofibrils were generally normal in the dendrites, even when these were diseased, and in the peripheral portions of the cells, but they were, as a rule, broken up in, or had disappeared from, the centre of the cells. Curious bladder-like appendages, into which neurofibrils could be traced, were attached to the bases of some of the cortical cells; they seem to have arisen by the central constriction of an elongated cell which thus becomes hour-glass shaped.

There was slight degeneration of the myelinated fibres of all parts of the forebrain, but it was in no place great. The pyramidal and cortico-pontine tracts were, on the other hand, considerably degenerated, the former especially in the cord. All the other systems of the brain-stem appeared practically normal when examined by the Weigert-Pal method. Marchi's method, on the other hand, revealed slight diffuse degeneration in all parts of the central nervous system, greatest in the course of the pyramidal and cortico-pontine tracts. The optic tracts appeared normal in both cases when stained by the Weigert-Pal method, but in one case there was recent degeneration visible by Marchi's method. The use of the specific neuroglia stain showed that the neuroglial proliferation was limited to the areas of the degenerating systems.

The eyes of the same two cases were examined microscopically, but unhappily the minute histology was complicated by the presence of post-mortem changes. In one eye of the one case, which was opened immediately after the autopsy, a minute hole was found at the fovea, but this could not be demonstrated in the other eyes which were examined in sections. There was degeneration of the ganglion cells and nerve fibres of the retina of both cases, but the cells of the nuclear layers remained intact. The changes which were visible in the ganglion cells were identical with those which have been described in the central nervous system; the cells were swollen, often vacuolated, the tigroid had disappeared, and the majority of the nuclei lay excentric. There was also evidence of œdema of the retina.



The explanation of the ophthalmoscopic appearances is probably that the white area around the fovea is due to cedema and the folding of the retina which results from it, while the dark red spot at the fovea is merely a contrast appearance.

The conclusions which have been drawn on the nature of the disease are that it is a primary cell affection, and that the initial change is disease of the interfibrillar protoplasm, and that the alterations of the neurofibrils are secondary to this. As regards its ætiology, there is no evidence of it being merely a condition of arrested development, and there is nothing to support the hypothesis that it is due to the action of toxins. It can therefore only be concluded that the degeneration is due to some inherent biochemical peculiarity of the protoplasm of the cell which is inherited.

GORDON HOLMES.

**A PECULIAR FORM OF AMAUROTIC FAMILY IDIOCY. (Ueber (315) eine besondere Form von familiärer amaurotischer Idiotie.)**

W. SPIELMEYER, *Neurolog. Centralbl.*, Jan. 16, 1906, S. 51.

THIS interesting communication records a form of disease which apparently has not been previously described. The four cases occurred in a family of five, the eldest member alone escaping. As the father contracted syphilis after the birth of the eldest child, it seems probable that the disease developed on a congenital syphilitic basis, though there was no clinical evidence or anatomical indication of this.

The symptoms began in each case at about the age of six years, with gradually progressive dementia, and diminution of vision due to retinitis pigmentosa. The blindness soon became complete in each case. There were no symptoms of palsy, and death occurred only from intercurrent illnesses, in each case at the age of puberty.

The disease is thus distinct from that which is known by the names of Waren-Tay and Sachs, though it has in common with it the symptoms of dementia and blindness.

The pathological conditions in the two cases which were examined were identical. They are referred to only briefly in the present paper, but will be published more fully elsewhere. There was universal cell affection throughout the whole central nervous system; the cells were swollen, the greater portion of their tigroid had disappeared, and they contained a curious granular deposit which stained by the fat dyes and frequently contained pigment. There was, on the other hand, relatively little change in the medullated fibres and axis-cylinders.

From the short account given, the pathological anatomy of this disease seems to resemble very closely that of amaurotic family idiocy.

GORDON HOLMES.

**AMAUROTIC FAMILY IDIOCY.** (Zur Kasuistik der Tay-  
(316) Sachs'schen Krankheit [Idiotismus familiaris amauroticus.]

W. STERLING, *Neurolog. Centralbl.*, Jan. 16, 1906, S. 55.

THIS paper contains the report of a typical case of amaurotic family idiocy. A child of Jewish parents came under observation at the age of eleven months, with the history that since the age of six months it had ceased to develop both mentally and physically, and had gradually become peevish and apathetic. There was no other case in the family.

On examination it was seen that the child was poorly developed and badly nourished and had well-marked signs of rickets. The functions of the cranial nerves were undisturbed and the pupils reacted briskly, though the child was undoubtedly blind and had been, according to the mother's account, from birth. Ophthalmoscopic examination revealed the characteristic cherry-red spot, surrounded by a white halo, in each macular region, and advanced optic atrophy. The muscles were small but hypertonic, especially those of the lower limbs. The latter were rarely moved voluntarily, and their strength seemed to be considerably diminished—they could not bear the child's weight. The arms and hands were stronger, and were almost constantly in apparently purposeless movement. The deep reflexes were increased, and plantar stimulation produced extensor responses of the great toes.

As in many of the other cases reported, there was marked hyperacusis; the child started violently at the slightest noise. A pronounced feature of the case was the less frequently observed symptom of automatic purposeless movements, especially those of sucking, swallowing, and yawning.

There was no pathological examination.

GORDON HOLMES.

**THE PORENCEPHALIC FORM OF INFANTILE PARALYSIS.**

(317) (Ueber die porenkephalische Form der zerebralen Kinderlähmung.) Dr A. DANNENBERGER, *Klinik f. psych. u. nerv. Krankheiten*, Bd. 1, H. 2. Halle a. S.: Carl Marhold, 1906.

THE author, after a preliminary description of porencephaly, its pathology and various ætiological factors, proceeds to a careful clinical description of four cases of this form of infantile cerebral

palsy, with in one of these an account of the post-mortem results. The first and the last of these showed clinically the characteristic symptomatological triad of imbecility, epilepsy, and spastic paralysis, with developmental arrest in the paralysed parts. The author, however, points out that according to the site of the cerebral lesion, one or all of these cardinal symptoms may be wanting, *e.g.* the spastic paralysis may be replaced by hemi-chorea, hemi-athetosis, or general chorea or athetosis. In the second case the diagnosis was open to question, the patient, a boy of eight years, having a markedly neuropathic heredity, but no history of birth trauma or post-natal convulsions, and exhibiting later moral rather than intellectual defect. The patient had no typical epileptic attacks, these being replaced by larval epilepsy in the shape of periodic attacks of excitement separated by quiet intervals, and in place of the spastic paralysis there was only slight comparative weakness of the muscles of the right side. The reflexes on the right side, though lively, were not pathologically increased. The diagnosis of porencephaly of slight degree in the neighbourhood of the motor centres—an admittedly fine diagnosis—is discussed minutely. The third case was one with marked convulsive epilepsy and hemiplegia, but with hardly any mental reduction; and the fourth, a clinically well-marked case, is of interest on account of the relation of the post-mortem findings, which are fully discussed, to the clinical picture.

R. CUNYNGHAM BROWN.

#### ON THE MILIARY DISSEMINATED FORM OF BRAIN SYPHILIS

(318) **AND ITS COMBINATION WITH GENERAL PARALYSIS.**

(Zur Lehre von der miliaren disseminierten Form der Hirnlues und ihrer Kombination mit der progressiven Paralyse.) E. STRÄUSSLER (of Prague), *Monatsschr. f. Psych. u. Neur.*, March 1906.

THE formation of miliary gummata is one of the rarest forms of syphilitic brain disease. In the cases previously recorded the gummatous formations usually occurred in the middle of a diffuse syphilitic infiltrative process, while two cases of miliary gummata on the ependyma of the lateral ventricles are on record. In the two cases here presented the gummata were situated in the deeper cortical layers, while the meninges covering the cerebrum showed no specific syphilitic changes; in one case there was a gummatous meningitis limited to the cerebellum, and in this region the gummata extended from the meninges into the brain substance itself. In both cases the cerebral cortex showed the histopathological features of general paralysis.

The first case was a man of 30, who, after a year's nervousness, became shortly before admission excited, talkative, full of plans. On admission he presented tremor of tongue, lively deep reflexes, slight deficiency of innervation of the left facial, but no speech nor pupillary defect; he was euphoric, expansive, restless; after five months' residence in hospital, during which his mood varied between exaltation and depression, he died after a series of epileptiform attacks. Microscopically the brain showed the characteristics of general paralysis. In addition there was in the cortex of the frontal and temporal regions a large number of miliary foci of a gummatous nature; these were in obvious relation to vessels, had irregular and ill-defined limits, and were most numerous in the deeper cortical layers. While in meningo-encephalitis the exudative process spreads in from the meninges, there was no such relation in this case. The origin of the miliary gumma consisted in a great infiltration of the vessel wall with cells of which the great majority were lymphocytes; obliteration of the lumen took place, the infiltrating cells and rod-cells (*Stäbchenzellen*) did not respect the vessel wall, but penetrated into the surrounding tissue, and regressive changes in the centre of the focus, with formation of giant-cells, occurred.

The second case was a man of 64, who, after a cataract operation three years previous to admission, began to show signs of mental enfeeblement; he became restless, irritable, disoriented, thought he was dead. His speech was drawling and scanning; deep reflexes were increased, there was double ankle clonus; his writing was unintelligible. He died from catarrhal pneumonia shortly after admission. The cortex presented the histopathological characteristics of general paralysis. In addition there was in the layers of the small and large pyramidal cells a number of disseminated foci. The foci in the previous case were always in relation to a vessel wall; in the second case there was no such relation, the focus consisting essentially of a central necrotic patch surrounded by an area of marked glia reaction, and in the later stages infiltrated with a large number of small round nuclei; no giant-cells were observed. It is interesting to note that in the central nervous system such a gummatous formation can arise without implication of the mesodermal tissue, the ectodermal tissue forming the basis of the new formation.

In both cases in the neighbourhood of the miliary gummata the paralytic process showed greater severity, as it may do in the neighbourhood of a glioma, or scar tissue.

Alzheimer, in discussing the histopathological diagnosis of general paralysis and brain syphilis, refers chiefly to syphilitic meningo-encephalitis. The above cases show the existence of a disseminated form of brain syphilis which is independent of the meningeal

changes, and where naturally the diagnostic points referred to by Alzheimer do not help. The cases reported are of little use for establishing diagnostic points, inasmuch as the picture was complicated by that of general paralysis. As to Nissl's view, that a meningo-myelitis invariably is found to accompany a meningo-encephalitis, the first case makes that statement doubtful, for a meningo-encephalitis of the cerebellum was present, while the medulla and upper cervical cord were free; the rest of the cord was not examined. With regard to the rod-cells of Nissl, these were extremely numerous in the first case, where the gummata were in relation to the vessel wall, and much more numerous in the neighbourhood of the gumma than elsewhere; this does not agree with Alzheimer's observation that these cells appear in a much more isolated manner in brain syphilis than in general paralysis. In conclusion, the author discusses certain relations of general paralysis to brain syphilis. The nature of the various foci is well illustrated in the plate which accompanies the article.

C. MACFIE CAMPBELL.

**REMARKS ON THE ÆTIOLOGY OF EPILEPSY.** (*Bemerkungen (319) zur Ätiologie der Epilepsie.*) EMIL REDLICH, *Wiener medicin. Wochenschr.*, 1906, Mai 26, S. 1074, u. Juni 2, S. 1147.

THE author follows Nothnagel in his division of the "causes" of epilepsy into predisposing and exciting. Treatment should aim at counteracting both these factors, though a practical difficulty is that the exciting causes are far from constant even in the same individual. A few of the less well known causes are dealt with by means of reference to cases observed in the past year. First, the relation of pregnancy, and especially labour, to epilepsy. Nerlinger has demonstrated that this connection has been unduly emphasised in the past because of the confusion existing between eclampsia and epilepsy, and that there is certainly no such thing as a special epilepsy of pregnancy. Nevertheless, it is not rare for this state to act as an exciting cause, particularly in women with special disposition—often hereditary. Numerous cases are quoted from the literature as evidence of this, and the experience of many authorities is referred to. Four cases are briefly described in which ætiological action of pregnancy seemed certain. Two of them revealed no other factor, two suffered psychical trauma at the end of pregnancy. Pregnancy only infrequently gives rise to epilepsy, because the required disposition is not present as a rule; but if this is there, pregnancy may light up epilepsy, just as may chorea, tetany, myelitis, disseminated sclerosis, etc. Two of the above cases

had suffered from convulsions in infancy, so that the exciting action of pregnancy was manifest. Whether the action is brought about through an infectious process, or *via* the effect on the circulation, or through metabolic changes causing the formation of the toxic products is quite unknown, though the first is not probable.

The question should abortion be induced arises in these cases, but the opinion of most authorities is that this should only be done if the status epilepticus be present.

A fifth case is described in which the attacks began four months after labour, and a few days after the passage of a tapeworm. The author discusses whether the labour, the presence of the tapeworm, or the use of a helminthintic is the operative factor. He holds that epilepsy may certainly be induced by the presence of tapeworms, particularly *tænia nana*, though in the case of *tænia solium* the possibility of an autogenous cysticercus in the brain must be considered. In a sixth case the author discusses whether a tapeworm was the cause of the attacks or whether the case entered into a special group of senile epilepsy characterised by mental disease, and described some six years ago.

Two further cases are related to illustrate the occurrence of peculiar attacks of timidity and strangeness during which the surroundings become suddenly quite unfamiliar. Gowers has previously described the same condition.

In conclusion, two cases are mentioned which raise the possibility of vaccination acting as an exciting cause. In both a secondary infection had taken place. Similar cases have been previously recorded.

ERNEST JONES.

#### ON NON-EPILEPTIC AFFECTIONS OF CONSCIOUSNESS OR

(320) **SHORT NARCOLEPTIC ATTACKS.** (Über die nicht epileptischen Absenzen oder kurzen narkoleptischen Anfälle.) M.

FRIEDMANN, *Deutsch. Arch. für Nervenheilkunde*, 1906, Bd. xxx., S. 462.

CASES are sometimes seen which differ from nerve giddiness in having no real disturbance of equilibrium, and from petit mal in the distinct maintenance of full consciousness; this however undergoes certain momentary alterations. Up to now these have been grouped, not as a separate condition, but as a variety of the affection called by Gélinau Narcolepsy, and previously described by Westphal under the title, "Peculiar Sleep States." It is now recognised that narcolepsy occurs, not only with larval epilepsy, but more often in cases of pure epilepsy and other functional

neuroses, such as hysteria and neurasthenia. The exact relation to epilepsy has been much discussed by Löwenfeld, Binswanger and other writers.

The author describes fifteen cases he has carefully studied personally, and nine he has discovered in the literature, and deals fully with the various problems arising in relation thereto. His chief conclusions are: these short narcoleptic attacks differ from ordinary petit mal in the incompleteness of the disturbance of consciousness, the tendency to occur under certain definite conditions, such as during meals, at work, after excitement, in sleep, etc. It also differs from epilepsy in producing no ill effects on either the body or mind. It seems to be commoner in the female sex. It practically never begins after thirty, and is not much rarer in children than in adults. In spite of its comparative innocuousness it is a very troublesome complaint on account of the frequency of the attacks. These are very constant in their symptomatology. The eyes turn upwards and remain fixed, the pupils dilated but reacting; the flow of thought is arrested, though consciousness is retained; the limbs are motionless and flaccid, or else may automatically continue the movement they were engaged upon; a sense of oppression is common in the attack; waking is usually quite complete, no symptoms remaining; an aura frequently occurs, especially the feeling that "it's coming again." Paralysis of the limbs is a rare symptom, occurring in only three of the fifteen cases. The duration of the attack is usually fifteen to thirty seconds, but not infrequently they last two or three minutes; in one adult, however, they used to last half an hour, and in one child two hours. The frequency of the attacks is their most variable feature, in one case it was as high as 100 times a day. It was curious to note that an intercurrent illness prevented any attacks while the patients were confined to bed. The most difficult question is that of the ætiology of the condition. Although severe sleep attacks may be due, as Ballet thinks, to bodily conditions, such as obesity, the slight ones are certainly psychical. The author's cases could be sharply divided into the two groups that Gélinau described, the primary idiopathic cases and the secondary symptomatic cases. Three each of children and adults of the present cases belonged to the former group; the rest were either neurasthenics or hysterics, more often the former. The prognosis is much better in the secondary cases, the total duration being as a rule from six to eighteen months. The primary cases are, however, far more obstinate, and one of those recorded had been in progress for fourteen years; exceptionally they may recover after a few years. As to the fear of epilepsy developing, that may be dismissed in the secondary cases, and in the primary ones it has been

recorded only once. Mental shock or distress almost always precedes the onset of the complaint. Heredity plays some share, especially in the primary form. A cousin of one of the author's cases suffered from the same condition, and in several of the recorded cases this was so with one of the patient's parents. Lastly as to the relation of the condition to allied causes of clouding of consciousness. Two fundamental features serve always to differentiate it—*first* the clouding affects only the higher functions of the brain, so that volition in thought is inhibited as in movement, but automatic movement may persist; *secondly* the attacks recur periodically in great numbers. There is a group of real sleep states, which may be called a second type of narcolepsy, that occurs also in neurasthenia, hysteria, or epilepsy. Again cases occasionally occur in which the inhibition is confined to thought, but voluntary movements can be carried out. Two such cases are described by the author. These three types of narcolepsy make it imperative to revise our present teaching, which usually attributes every uncaused disturbance of consciousness to epilepsy. The attacks now described may also be confounded with the sudden "refusals to think" that arteriosclerotics sometimes suffer from, but these last many minutes or even an hour.

ERNEST JONES.

**PUERPERAL ECLAMPSIA AND PARATHYROID INSUFFICIENCY.**

(321) G. VASSALE, *Società Medico-Chirurgica di Modena*, Meeting of 4th July 1906.

THE parathyroid theory of puerperal eclampsia, formulated last year by the author, on the ground of experimental and clinical observations, has since received confirmation — (1) from pathological observations which have served to demonstrate in persons who have died from eclampsia morbid changes in, or congenital absence of, one or two parathyroid glands (Pepere, Zanfognini); (2) from additional clinical observations, showing the beneficial effects of parathyroid treatment in eclamptic convulsions (Zanfognini, Stradivari); and (3) from new experimental researches carried out upon gravid cats and rats (Zanfognini, Erdheim, Thaler, and Adler), which have confirmed the conclusion that in latent parathyroid insufficiency there constantly occur in the last third of pregnancy severe parathyreoprival convulsive phenomena (experimental eclampsia). The author gives an account of the history of three gravid bitches, from each of which he this year removed three parathyroid glands. The animals remained well until the last few days of pregnancy. In two of them experimental eclampsia developed about two days before parturition. To



one of these animals parathyroidin was given by mouth in very large doses. The convulsions ceased, and under the influence of parathyroidin, the administration of which by the mouth was continued after the cessation of the first convulsive seizure, the bitch, without suffering from any further convulsion, gave birth to three pups, which died within three days owing to want of milk in the breasts of the mother. The second bitch was not given parathyroid treatment, and about forty hours after the onset of the first slight attack of tetania parathyreopriva, it died in a violent convulsive seizure, without having expelled the fetuses, although these had reached their full term. In the case of the third bitch, convulsions occurred only a few moments before parturition. A large dose of parathyroidin was administered, and the animal succeeded in giving birth to six pups, four of which it suckled and reared. In the course of lactation it again had a violent seizure of tetania parathyreopriva, which was combated by large doses of parathyroidin. Subsequent to this it remained, like the first bitch, in good health. The urine of these animals contained albumen ( $\cdot 05$  to  $\cdot 1$  per cent.), the amount of which kept increasing during the last days of pregnancy. The albumen disappeared fairly rapidly after parturition. The onset of experimental eclampsia in these animals was preceded by a period of oliguria and anuria. The author also points out the clinical analogy that exists between the disorders of the renal functions in these animals and those that are to be observed in eclamptic women. Concerned with the pathogenesis of the renal disorders of pregnancy, which are of chief importance as determining causes of the manifestation of a latent parathyroid insufficiency, and, therefore, of the onset of eclamptic convulsions, there are not only autotoxic causes, but also mechanical causes (compression by the gravid uterus), which induce disturbances in the renal blood circulation and urinary stasis. The author has found that partial occlusion of the ureters of dogs, upon which a partial parathyroidectomy has been performed, determines the rapid development of severe parathyreoprival convulsions, which result in the death of the animal in from fifteen to twenty hours. It is known that muscular fatigue and nervous exhaustion are also capable of determining the occurrence of symptoms of parathyroid insufficiency. It is thus easy to understand how in primiparæ, in whom the mechanical causes, dependent upon compression by the gravid uterus, are undoubtedly of greater importance, and in whom also the duration of the labour is longer, eclampsia occurs more commonly than in multiparæ.

AUTHOR'S ABSTRACT.

**PRELIMINARY REPORT OF THE TREATMENT OF IDIOPATHIC  
(322) EPILEPSY BY APPENDICOSTOMY FOR COLONIC IRRIGATION.** LA PLACE (Philadelphia, Penn.), *Journ. Am. Med. Assoc.*, June 2, 1906.

EPILEPSY is but a symptom revealing the reaction of the general nervous system to some irritation, either as a result of pressure from injury, or as a result of the accumulation of some toxine.

In the treatment of numerous cases of so-called Idiopathic Epilepsy, it has been found that the establishment of an intelligent diet is one of the most important elements.

Metchnikoff states that the colon is a receptacle for refuse undigested matter; that human life could be sustained in a more physiological state without its presence.

That the power of absorption by the colon is remarkable may be shown by the length of time during which patients may be nourished from rectal feeding alone.

It is reasonable to suppose that from this colonic reservoir toxic material is absorbed, especially in cases where chronic constipation exists. Any method, then, which would destroy this toxine, or prevent its absorption, would contribute toward lessening the nature and frequency of epileptic attacks. With this theory in view, La Place has created an artificial fistula at the beginning of the colon, and directed the patient to flush out his colon with two gallons of warm water morning and night. Four cases of similar nature have been treated in this way, and have shown distinct improvement—i.e. lessening number of convulsions, causing a more cheerful appearance, and an improvement in appetite. They have not been operated upon for a long enough time to be reported in full at present.

C. H. HOLMES.

**CESSATION OF THE PULSE DURING THE ONSET OF EPILEPTIC  
(323) FITS.** A. E. RUSSELL, M.D., *Lancet*, July 21, 1906, p. 152.

THE author describes a case of cessation of the radial pulse at the onset of epileptic fits for three-quarters of a minute or more. He notes other cases where the same phenomenon has been observed, and suggests that the cardiac arrest may be the cause of the fits through the production of cerebral anæmia.

While not attempting to explain *all* epileptic fits in this way, the author thinks that such arrest may be much commoner than is suspected. If observations were made on the pulse at the onset of fits by those whose work brings them into contact with epileptic patients in considerable numbers, it would soon be established whether such cardiac arrest is of occasional or of frequent occurrence.

W. B. DRUMMOND.

**ON HYPERÆSTHESIA OF THE VISUAL PERIPHERY.** (Über  
(324) *Hyperaesthesie der peripherischen Gesichtsfeldpartien.*) A.  
PICK, *Neurolog. Centralbl.*, June 1, 1906, p. 498.

THE author contrasts the careful studies that have been made on hypoesthesia of the retinal periphery with the absence of interest displayed in the reverse condition. He refers to a previous communication of his (*Brain*, 1903) describing a patient in whom hyperaesthesia of the retina was due to a pathological prolongation of the visual impressions. It is known that under normal circumstances attention given to a central object fades gradually into that given to an object in the periphery; and further, that men may be divided into two types, according to whether they make much or little use of their visual periphery. William James says that women use it more than men. The ease with which the eye can be focussed so as to bring an important object opposite to the macula is also of course important in this connection. When the focussing movements are defective, the effort to retain both the directly seen and the indirectly seen objects in the field leads, as Hering has shown, to a doubling or dispersion of attention so that the central image reaches consciousness with an effort that is translated as pain. The author mentions the case of a neurasthenic who suffered from obsessions and impulsions, and who complained of a peculiar extreme visual trouble, saying, "Too much streams into my eye." On closer examination it was noticed that in fact he did see too much, in that he had a clear perception of many objects besides the one looked at directly; this caused disturbances of attention, as above described, so that the central vision was disturbed, resulting in pain from the antagonism of the two sets of images. To the perimeter the field appeared normal. In another instance, the patient being a peasant girl, the visual trouble was the starting-point of a severe psychopathic disturbance. She was extremely shy and timid, and on going to Vienna soon became greatly distressed by the turmoil of the city, particularly the electric trams, of which she was terrified. She gave up work and withdrew into herself. Her visual symptoms were as follows: when anyone moved on her side he became clearly seen as though he occupied the centre of the field. This naturally gave her the feeling of squinting towards him, especially as the same thing might occur simultaneously on both sides. No objective movements were visible; on these occasions, indeed, the eyes were motionless under conditions when a lateral movement was expected. This phenomenon occurred only in an outward or downward direction, not upwards. The field of vision, repeatedly examined with the perimeter, was remarkably extensive for both white and colours. The diagnosis of an obsessive im-

pulsion was negatived by the coincident divergence of the eyes, by the condition occurring only with moving objects, and by its intensity increasing with the number of moving objects.

Pick's conclusion is that the varying æsthesia of the different parts of the retina is a protective mechanism whereby the peripheral objects are excluded to a greater or less degree. This capacity is only gradually developed, and is not present in children when their lateral vision begins, at the fifth month.

ERNEST JONES.

**THE PUPIL REFLEXES IN MITRAL VALVE LESIONS. (Des (325) réflexes pupillaires dans les cardiopathies mitrales.)**

BRAILLON, *Gazette des Hôpitaux*, June 21, 1906, p. 831.

THE author first refers to the syndrome, first pointed out by Babinski and confirmed on all sides since, consisting in the co-existence of the Argyll-Robertson sign and a lesion of the aorta. By its means we are now able frequently to decide whether a given aortic lesion is due to syphilis or to some other cause, such as traumatism. It may be regarded as established that when, through an affection of the centrifugal path, the reaction to light is abolished, with or without the reaction to accommodation, we may be quite certain that a chronic syphilitic meningitis is present. This has been confirmed in a striking manner by the study of the cerebro-spinal fluid in such cases. The author has recently observed two cases which raise the question of the syphilitic origin of certain mitral affections. *Obs. I.* Patient was a man aged 66. He had double hydrothorax, large, painful liver, œdema of the lower limbs, rapid, soft pulse, scanty urine, hypertrophied left ventricle, with the classical signs of organic mitral regurgitation. The pupils were myotic, reacted feebly to accommodation and not at all to light. No other signs on examining the nervous system. He had had a hard chancre and secondaries at the age of twenty-seven. *Obs. II.* Patient was a man of 45. Rapid, feeble pulse, hypertrophy and dilatation of the left ventricle, with signs of organic mitral regurgitation. There were evidences of old interstitial keratitis but not of iritis. The pupils were of average size and regular; they responded feebly to accommodation, but not at all to light. He had had syphilis badly ten years previously. His wife was well, but one of her pupils was dilated and responded neither to light nor to accommodation; a study of the consensual reflexes showed the integrity of the centripetal path.

Although the cases were not examined after death, there was no doubt of the diagnosis of an affection of the mitral valve. Syphilis was probably the cause of this affection, there being no

indication in the history of any other factor that might account for it. The author's previous researches have shown that Koch's bacillus may undoubtedly give rise to tuberculosis of the endocardium without any characteristic anatomical appearance of this, so that the specificity of the structure of the endocardium may mask the specificity of the germ. This may also be the case with Schaudinn's spirochæte, and syphilis may be the cause of many cases of mitral affection that are now unexplained. Albespy's observations on the frequency of mitral lesions in tabetics support this suggestion.

ERNEST JONES.

**THE ABDOMINAL REFLEX IN ENTERIC FEVER.** J. D. ROLLE-  
(326) STON, *Brain*, Spring 1906, pp. 99-111.

THE paper is based on the study of sixty patients who were admitted to hospital certified to be suffering from enteric fever. In forty-five cases this diagnosis was confirmed; the remaining patients were found to have other diseases. In the former the abdominal reflex was affected to a varying degree in forty-two (93·3 per cent.), it was completely lost in thirty-one (68·8 per cent.), impaired in a greater or less degree short of absolute extinction in eleven cases (22·2 per cent.), unaffected in three cases only.

In only three of the fifteen patients who were not suffering from enteric fever was the reflex affected. The first was a case of suppurative appendicitis, the second one of acute cancerous peritonitis, and the third was that of a woman, aged 54, suffering from lobar pneumonia, in whom the absence of the reflex was regarded as the result of her age and wrinkled condition of the abdominal wall. The examination should be made daily throughout the disease, as a single negative result is of little value. The abdominal muscles must be relaxed. Ticklish and apprehensive patients should have their attention diverted by conversation or other means.

The best method to elicit the reflex is lightly and rapidly to stroke the skin of the abdomen with the end of a penholder. The author adopts Oppenheim's division of the reflex into a supra-umbilical and infra-umbilical zone. In enteric fever the infra-umbilical reflex is the first to disappear and the last to return. The supra-umbilical reflex may remain active throughout the disease, or, as more commonly happens, a slight response rapidly exhausted after a few stimuli may be obtained in the epigastrium or sides of the abdomen alone, when stimulation of the rest of the abdomen provokes no response.

The author's conclusions are as follows :—

1. The abdominal reflex is affected in a very large number of cases of enteric fever, the percentage of cases in which it is entirely lost exceeding those in which its normal activity is diminished only.

2. From its absence under 50 being confined to certain nervous diseases and acute abdominal conditions, notably appendicitis and enteric fever, the absence of the abdominal reflex in a given case of continued pyrexia in any patient below 50 is of considerable diagnostic value.

3. The comparatively transient nature of the affection of the abdominal reflex in enteric fever is a striking contrast to the more chronic affection of the knee- and ankle-jerks in diseases associated with peripheral neuritis, *e.g.* diphtheria.

4. Return of a lost reflex, and *a fortiori* resumption of its normal activity, are a valuable indication of commencing convalescence, and often correspond with lysis and characteristic changes in the fæces and urine.

5. The objective sign of return of the reflex is often associated with the return of the subjective feeling of ticklishness normal to the individual.

6. In reappearance of pyrexia in convalescence, the condition of the abdominal reflex is a valuable index of the nature of the pyrexia.

7. No constant relation exists between the condition of the abdominal reflex and that of the tendon reflexes.

8. The frequency, degree, and duration of impairment of the abdominal reflex are, as a rule, in direct proportion to the age of the patient.

AUTHOR'S ABSTRACT.

#### **ATROPHY OF THE GLANDS AT THE BASE OF THE TONGUE AS**

(327) **A SIGN OF SYPHILIS.** N. B. POTTER, *Boston Med. and Surg.*

*Journal*, March 8, 1906.

IN a study of three hundred cases of various kinds this symptom was present in about one-half of the patients who exhibited reasonable evidence of previous syphilis, and in only about ten per cent. of cases without satisfactory evidence of previous syphilis. Examination by palpation is more trustworthy than by vision.

The tongue, after being protruded as far as possible, is grasped and held by a hand covered with a towel. The region behind the circumvallate papillæ is explored by the index finger of the other hand, introduced along the dorsum of the tongue. The conclusions are, that when the papillary glands at the base of the tongue are normal, syphilis may be excluded, while typical atrophy of these

glands in an individual below the age of fifty is indicative of syphilis. A moderate or slight degree of atrophy is of little diagnostic importance.  
C. H. HOLMES.

**PSEUDO-BULBAR PALSY IN A CHILD.** (*Paralysie pseudobulbaire* (328) *chez un enfant.*) RAYMOND and LEJONNE (*Soc. de Neur. de Paris*), *Rev. Neurol.*, April 5, 1906.

THE case concerns a boy of 11, who was in perfect health up to one year ago. His symptoms commenced by slight weakness of the left leg, followed in a month or two by diarrhoea and spasmodic laughter, faulty articulation, maladresse of hands, specially on the right. In March 1906 he presented a double hemiplegia, his movements being stiff and awkward, but Babinski's sign was absent. There were in addition double facial palsy, more pronounced on the left side, complete paralysis of the tongue, involvement of the motor fifth, but no palsy of the larynx, and practically no difficulty in swallowing.

The authors think the condition must be supranuclear, i.e. pseudo-bulbar, because while the tongue is paralysed, it is not in the slightest degree atrophied; there are no changes in the musculature of the body or face, no fibrillary contractions, and no change in electrical excitability. They do not commit themselves as to its cause.  
S. A. K. WILSON.

**A CASE OF TIC.** (*Iconographie de l'évolution d'un cas de maladie des* (329) *tics.*) ROUBINOVITCH, *Nouv. Icon. de la Salpêtrière*, mars-avril 1906.

THIS paper consists of a brief account of the evolution of certain tics in a young man twenty-three years old. It is illustrated by a series of excellent photographs illustrating the convulsive movements of the patient.  
S. A. K. WILSON.

**INTERMITTENT CLAUDICATION OF THE SPINAL CORD.** (330) (*Sur la claudication intermittente de la moelle épinière.*) DÉJÉRINE, *Rev. Neur.*, April 30, 1906, p. 341.

THREE cases are quoted of healthy individuals in the prime of life who are afflicted with intermittent paralysis of one or both legs. In repose there is no inconvenience, and in two cases where the symptoms are unilateral, the subjects are not conscious of any difference between the two legs as far as the muscular power is concerned. It is only after walking a certain length that they

find one or both lower extremities becoming heavy and progressively heavier, more and more difficult to move, till soon they are incapable of making any movement at all. A rest of a few minutes suffices for the phenomenon to disappear, and for their natural power and suppleness to return to the paralysed limbs. If the patient be examined when in the paralysed state, it will be found that the reflectivity of his lower limbs is greatly increased, and that sometimes an actual extensor response is present.

There can be no doubt that the symptoms are analogous to those of intermittent claudication from arterial disease, because the pain and weakness occur after muscular exertion or because the condition disappears with rest; and the facts that there is no obliteration of the peripheral pulse in the legs, and no cyanosis or coldness of the skin, are of localising value. The disease may be either peripheral or medullary, but the absence of the latter symptoms shows it must be medullary in the cases quoted. In the great majority of cases syphilis is the cause of the condition, and antispecific treatment is urgently called for. The physiological pathology indicates that the process is one of *meiopraxia* of part of the spinal cord, that is to say, insufficient irrigation by the blood stream. The ischæmia produced by exertion reveals itself in functional insufficiency.

S. A. K. WILSON.

#### INTERMITTENT CLAUDICATION OF NERVOUS CENTRES.

(331) (*La claudication intermittente des centres nerveux.*) GRASSET, *Rev. Neur.*, May 30, 1906, p. 433.

IN this interesting communication Grasset refers to the article of Déjérine on the same subject in an earlier number of the *Revue Neurologique*. He shows that strictly speaking Déjérine ought to have called his paper "intermittent claudication of the *anterior* (antero-lateral) part of the spinal cord," for reasons readily appreciated.

Grasset emphasises the widespread nature of the lesion that may cause intermittent claudication, and the possibility of its occurrence in cerebrum and brain and spinal cord. In the first case, it reveals itself chiefly by transient amnesia, intellectual fatigue, and aphasia, often by very incomplete "strokes," momentary loss of consciousness, of orientation, etc. Intermittent claudication of the mesencephalon is seen in Cheyne-Stokes respiration, paroxysmal and transient, in vertigo, which may pass into the syndrome of Stokes-Adams, with epileptiform attacks and bradycardia, etc. In the spinal cord there may be intermittent claudication of the posterior cord, indicated by paroxysmal painful feelings of constriction round the trunk, associated with pseudo-angina and gastralgia, and possibly akin to the gastric



crisis of tabetics. The colic of arterio-sclerotics is almost certainly due to intermittent claudication.

Finally, one may have the ordinary peripheral (muscular) intermittent claudication, the symptomatology of which is familiar. Transient paræsthesias and crises of acroparæsthesia are probably to be attributed to this condition.

Grasset considers many of the symptoms of tabes have a similar explanation.  
S. A. K. WILSON.

**A CASE OF TACTILE APHASIA.** (*Un cas d'aphasie tactile.*)  
(332) RAYMOND and EGGER (Soc. de Neur. de Paris), *Rev. Neurol.*,  
April 5, 1906.

THE patient was a woman 61 years old, with a right brachial monoplegia of four months' duration. There was perfect conservation of all forms of cutaneous sensation over the palmar aspect of the hand, but there seemed to be fairly constant post-axial localisation when topognosis was tested. There was no hemianopsia. There was no aphasia, and only occasionally some verbal amnesia—muscular sense was conserved in its entirety in the right hand.

When various objects were placed in the right hand, the patient recognised their forms well enough, and could say whether they were round or square, thin or thick, large or small, rough or smooth, etc. Yet in each instance she failed to name the object she was holding, whereas the moment she had it in her left hand she could tell what it was. An orange was described as big, round, hard, rough, but she could get no further.

The condition is therefore one of pure tactile aphasia, analogous to word deafness; in the latter case the patient hears perfectly what is said, but the acoustic impressions do not evoke the images of the words.

The patient in this instance is not suffering from astereognosis, for she recognises the forms of objects, and is not therefore to be classified with the cases of *Tastlähmung* described by Wernicke, since these were not only unable to recognise objects by touch, but were astereognostic as well.  
S. A. K. WILSON.

**CONTRIBUTION TO THE LOCALISATION OF THE MUSICAL  
(333) TALENT IN THE BRAIN AND ON THE SKULL.** (*Beitrag zur  
Lokalisation des musikalischen Talentos im Gehirn und am  
Schädel.*) AUERBACH, *Archiv. f. Anat. u. Phys.*, 1906, p. 197.

IN this article the writer gives an account of the literature bearing upon this intricate question, records certain observations of his own,

and suggests a series of headings under which future investigations might be recorded.

Among the most important sets of records were those of Retzius, who was inclined to associate the mathematical talent with a strong development of the parietal region, particularly of the angular gyrus. The brain of Gylden the astronomer, that of Loven the histologist, and that of Helmholtz, who all possessed great musical gifts, showed a special development of this part and of the first temporal convolution. A similar condition was found in the brain of the violin-virtuoso Rudolf Lenz. In the case of Naret Koning, first concert-master in the Frankfurt opera, who possessed an extraordinary ear for music, with great musical judgment, and whose brain was carefully examined by the writer, the hinder part of the left upper temporal convolution and supramarginal gyrus were strikingly developed. Further, the writer believes that he can observe in musically-gifted people a special development and roundness of the skull in the temporal region. He gives eight points to which he thinks future investigators might direct attention, as well as copious references.

JOHN D. COMRIE.

## PSYCHIATRY.

**THE INVESTIGATION OF THE TRAUMATIC NEUROSES BY**  
 (334) **PSYCHO-PHYSICAL METHODS.** (*Die Untersuchung von*  
*Unfallnervenkranken mit psycho - physischen Methoden.*)  
 Oberarzt CURT V. LEUPOLDT, *Klinik f. psych. u. nerv. Krankheiten*,  
 Bd. 1, H. 2. Halle a. S. : Carl Marhold, 1906.

IN the whole field of neurology there are few, if any, diseases which occasion more trouble and give rise to more controversy than the traumatic neuroses. Unfortunately these controversies are fought out in the full light of day, in legal courts, from which the medical witnesses have not always emerged with credit. Almost invariably in these vexatious cases the symptoms offered by the patients claiming damages are altogether subjective in character, and for this reason the results of minute psycho-physical methods of examination, such as are in operation at Giessen under Prof. Sommer, will, in time, it is to be hoped, furnish objective and satisfactory data on which to found an accurate diagnosis. That this has not yet been attained is evident from a perusal of Dr Leupoldt's careful description of the investigation made in nine cases of this nature. The methods employed include, in addition to ordinary neurological examination, the application of Sommer's apparatus for the three-dimensional registration of

movements to the attitude of the body and its members, to tremors of the hand and finger, and to "fright-reaction" on sudden noises; ergographic results with Mosso's instrument; time reactions; plethysmographic investigations and, on the intellectual side, tests in reckoning, in word memory according to Ranschburg's plan, in verbal association, and so on. Each of Dr Leupoldt's cases was exhaustively analysed and the results are given in extenso in this paper. In none of these cases does it appear that the tests employed did more than corroborate a diagnosis previously arrived at by ordinary methods, except in one case, in which the patient had been considered a simulator, but after examination was considered to be affected by "a particular kind of mental inhibition" giving rise to delay in all reactions and, apparently, incapacity for labour. No more definite diagnosis is given in this case, and here, as in the others, an inherent difficulty is presented by the impossibility of separating incapacity from disinclination, that is, as Dr Leupoldt admits, the subjective elements cannot be discounted even in these tests.

R. CUNYNGHAM BROWN.

**ON THE PUERPERAL PSYCHOSES.** (*Ein Beitrag zur Lehre* (335) *der Puerperalpsychosen.*) A. MÜNZER (of Heidelberg), *Monatsschr. f. Psych. u. Neur.*, April 1906.

THE author gives the result of his analysis of 101 cases where a psychosis developed in relation to gestation, the puerperium, or lactation; the term puerperal psychosis is applied widely to psychoses arising during any of these periods. The percentage of puerperal psychoses, estimated in relation to all female admissions, was 8 per cent., but in relation to the number of patients who had borne children was 21 per cent. The occurrence of the psychoses in relation to the three main periods was 19 during pregnancy, 56 in the lying-in period, and 26 during lactation. In discussing the ætiological factors, Münzer agrees with Schmidt, who lays great stress on the physical exhaustion due directly to child-birth. During pregnancy the placental circulation is a disturbing element in the general economy of the system. The fact that many authors lay great stress on the element of infection is in part explained by the frequent occurrence of fever, which, however, is frequently the only symptom to suggest infection.

In his series the author found that 56 cases occurred between the ages of twenty and thirty; the usual period given by authors as most common is between thirty and thirty-five. Pregnancy psychoses occurred on the average at a later period than the other forms.

Puerperal psychoses presented a larger percentage of cases

with bad heredity than other psychoses; the other ætiological factors were numerous and varied.

As to the nature of the psychoses, 26 were manic-depressive insanity, 53 were dementia præcox, dementia paralytica occurred 6 times, amentia (acute hallucinatory confusion) also 6, hysteria furnished 1 case, while in 9 the diagnosis was doubtful. The cases of dementia præcox were usually of the catatonic form, and presented frequently marked depressions. The psychoses in puerperal cases presented the same features as in the non-puerperal cases. The amentia presented the identical features which are found in cases occurring after other exhausting causes.

The author concludes that we do not know as yet a specific puerperal insanity, but it is not impossible that further analyses may discover features special to puerperal cases; the predominance of depressions during pregnancy is referred to in this relation.

As to prognosis, the cases occurring in pregnancy were the gravest. The prognosis of puerperal psychoses is the prognosis of the same psychosis occurring in relation to other factors.

C. MACFIE CAMPBELL.

**ON THE WANT OF INSIGHT OF ALCOHOLICS.** (Zur Lehre von (336) der Einsichtiglosigkeit der Alkoholisten.) O. JULIUSBURGER (of Steglitz), *Monatsschr. f. Psych. u. Neur.*, Feb. 1906.

IN this article the author discusses the grounds of the frequent inability of alcoholics to recognise the real ætiological factor in their mental disorder. He gives briefly in 25 cases the amount of money spent in drink, the wages and rent of the patient, and the reasons given by the latter for his drinking habits. The importance of the social atmosphere in determining the latter is most important, and cannot be neglected in any attempt at a permanent cure; the traditions of the classes from which the alcoholics are chiefly recruited must be known and combated if prophylaxis is to be really serious. In view of these factors Juliusburger emphasises the importance of a purposeful ethical treatment during detention: the apparent weakness of the alcoholic has frequently a sociological rather than an individual explanation.

C. MACFIE CAMPBELL.

**MIXED CONDITIONS IN EPILEPSY AND ALCOHOLISM.** (337) (Mischzustände bei Epilepsie und Alkoholismus.) Dr F. CHOTZEN (of Breslau), *Centralbl. f. Nervenheilk. u. Psych.*, Feb. 15, 1906.

THE close relationship of epilepsy and alcoholism is well known; the abuse of alcohol may lead to epileptic attacks, and epilepsy

with its symptoms and constitution predispose to alcoholism. Post-epileptic deliria resemble alcoholic deliria, but the well-marked affect, the fantastic ideas of persecution, the prominence of hypochondriacal ideas, the tendency to religious delusions, and the variable disorientation, are typical of the pure epileptic delirium. These may, however, be absent, and one may, on the other hand, in an epileptic meet a simple occupation delirium with tremor and complete disorientation. As a rule, the epileptic delirium presents a more varied and changeable picture than the alcoholic. The author has not seen in a non-alcoholic epileptic a typical alcoholic picture, but in alcoholics the epileptic attack may be the precipitating cause of a delirium presenting the pure picture of the alcoholic delirium. In alcoholic epileptics an acute hallucinatory condition without the dream-like state of consciousness of the epileptic, and without the fantastic, anxious delusions, may be met with. Such a picture frequently takes on, in its later course, the epileptic features. In one case, neither convulsions nor other epileptic symptoms had been observed. In cases of recurrent alcoholic attacks with the later development of epilepsy, the later alcoholic attacks may gradually assume an epileptic colouring. The anxiety may become prominent, the orientation extremely variable, with sudden misinterpretation of environment, abrupt assaults, great irritability, delusions of a religious content, hypochondriacal delusions. The epilepsy, as well as the deterioration after successive alcoholic attacks, is due to the association of arterio-sclerosis. The author discusses the question whether certain motor phenomena of a catatonic nature, occasionally met with in alcoholic psychoses, may not in reality be epileptic symptoms. He records one case of an alcoholic who, after several convulsions, showed a stupor with negativism, mutism, abrupt and unexplained actions, with dulling of the sensorium and absence of the pupil reflex; there was no affect observed; the epileptic nature of the stupor was seen in the general dulling and the absence of pupil reflex.

In the second case which he reports, the stupor preceded the development of epileptic attacks, and the latter were followed by a delirium. The behaviour of the patient during the stupor was that of a catatonic.

In a third case, in which nothing in the history indicated previous epilepsy, the patient had a delirium with hallucinations, chiefly auditory; hypochondriacal feelings of varied nature followed, and two days later he showed mutism, sat in stereotyped attitudes, showed *flexibilitas cerea*. This gave way to the previous picture, and in nine days patient quieted down, became quite clear and had insight.

In a fourth case the course of the psychosis was that of the

alcoholic delirium, although the patient in the early stages had presented a catatonic picture.

In depressive alcoholic psychoses complicated with motor phenomena, the possibility of a mixed condition with epileptic features cannot be excluded, and the author suggests a similar way of regarding several severe psychoses in marked alcoholics and in traumatic cases.

C. MACFIE CAMPBELL.

**PSYCHIC AND MOTOR DISTURBANCES CAUSED BY ALCOHOL, (338) IN PARTICULAR AMONGST THE NEUROTIC.** (Ueber Bewusstseinsveränderungen und Bewegungsstörungen durch Alkohol besonders bei Nervösen.) Privatdozent Dr DANNE-MANN, *Klinik. f. psych. u. nerv. Krankheiten*, Bd. 1, H. 2. Halle a. S.: Carl Marhold, 1906.

UNDER the above rather high-sounding title, Dr Dannemann describes the case of a man sentenced in 1903 to five years penal servitude for murder. Briefly the facts relating to his crime are as follows. The prisoner, æt. 21, whilst partially intoxicated, became involved in a quarrel and received a blow on the nose and had his ears boxed by a certain S. This was at 10 in the evening and was soon settled peaceably, prisoner and many others proceeding to a convenient tavern, without, however, S. the aggressor. About midnight the prisoner, not obviously drunk, went home and, passing on his way the home of S., became suddenly inflamed with anger, hastened home, seized a gun and ran back to the tavern. Here, in a room crowded with guests, but from which S. was absent, he discharged his weapon at an unoffending man, apparently under the impression that he was S., wounding him fatally, then fired again, wounding another who afterwards succumbed, and injuring several others. He was placed by the authorities under mental observation, and being found neither insane nor weak-minded, was sentenced as above. In investigating the case, Dr Dannemann obtained from his parents a criminal family history, his father and his brother having been frequently convicted of violent assault, but no heredity of insanity, epilepsy, or other nervous disease. The prisoner was stated to have been subject all his life to sudden accessions of anger, to have stayed out all night and wandered for no reason at times, and even up to a few months before his crime to have wet his bed. In 1900 he suffered two severe head injuries followed by brief unconsciousness. Also, a few weeks before incarceration, he had violently assaulted another man in a fit of rage following a quarrel—in this case also a late reaction. Under observation he was found to be mentally well endowed, normal in emotional reactions, and apparently of good moral character.

Memory for past events was good and also, when first examined, for the events leading up to and during his homicidal acts. Later his memory for these last events became confused and his evidence contradictory. During the whole period of his observation he showed no special characteristics of epilepsy, but during the last few weeks his sleep became disturbed, he suffered from headache, and was found to be markedly intolerant of alcohol. Tested by Sommer's apparatus, marked tremors were observed even after moderate doses, much more pronounced than in other normal individuals after prolonged drinking. Also, after comparatively small doses of alcohol, his power of observation and attention was proved to be unusually deficient. On the grounds of these observations, Dr Dannemann concludes that the prisoner was an individual of excitable temperament, psychically abnormal, and most probably the sufferer from *epilepsie larvée*. Prof. Aschaffenberg has shown the high percentage of cases of epilepsy exhibiting alcoholic intolerance, but until it is proved that this is not shared by any normal individuals, too much stress should not be laid on this sign, and Dr Dannemann's hypothesis, though suggestive, is somewhat unconvincing. R. CUNYNGHAM BROWN.

**ON THE EXPERT VIEW OF CRIMES COMMITTED DURING  
(339) INTOXICATION.** (*Zur gerichtsärztlichen Beurteilung der im  
Rausche begangenen Verbrechen.*) R. GAUPP (of Munich),  
*Centralbl. f. Nervenheilk. u. Psych.*, Feb. 1, 1906.

THIS article is a criticism of the views published by Hoppe in the January number of the *Centralblatt*. Gaupp admits the scientific truth of Hoppe's attitude towards the phenomena of intoxication and chronic alcoholism; he joins issue with him on his practical conclusions. It is the duty of the expert as such to aid in the carrying out of the laws in the spirit in which they were framed, and not to run counter to that spirit.

The law has not included intoxication as one of the causes which reduce the responsibility of the individual, and to put forward intoxication as removing the responsibility of the individual on the same footing as other conditions of mental disorder would be to fail to interpret the existing law and would be to expose the expert to ridicule. The majority of serious crimes are committed under the influence of drink. It is impossible to dismiss scot-free such individuals; there are not sufficient institutions in which they could be treated; the only practical solution under present conditions frequently is to subject them to imprisonment.

C. MACFIE CAMPBELL.

**THE FORENSIC OPINION AND TREATMENT OF CRIMES  
(340) COMMITTED BY THE INTOXICATED AND THE CHRONIC  
ALCOHOLIC.** (Die forensische Beurteilung und Behandlung  
der von Trunkenen und von Trinkern begangenen Delikte.)  
H. HOPPE (of Königsberg), *Centralbl. f. Nervenheilk. u. Psych.*,  
Jan. 5, 1906.

HOPPE reviews the laws of various countries regarding crimes committed by individuals either intoxicated or showing the result of alcoholic deterioration. He insists upon the fact that alcoholic intoxication is essentially a transitory mental disorder, and concludes that the responsibility of the individual should be determined in view of this fact. It is not sufficient to say that the individual is responsible for causing this mental disorder—social influences are a potent factor, and the reactions of various individuals differ. Cramer and Heilbronner recommend that a physician should decline to give an expert opinion on the responsibility of an intoxicated person, unless the individual has presented morbid symptoms. Hoppe looks upon this as a failure to accept one's scientific responsibility; the physician should insist on the essentially pathological condition of the intoxicated. He maintains a similar attitude with regard to the criminal acts of chronic inebriates. Punishment in these cases is absolutely fruitless: the prison is a quite ineffective means of answering the problem. The author proposes that where a man with a previously good record has committed a crime under the influence of alcohol, which he would not have done when sober, he should be conditionally released if an expert declare him irresponsible at the time of the crime, but that he should be bound over to keep the pledge; and that in the event of his failure to do this, and especially if he should again commit a misdemeanour under the influence of drink, he should be liable to serve the sentence for the first offence as well. Repeated misdemeanours should make him liable to be sent to an institution for inebriates for an adequate period, at least for one or two years.

C. MACFIE CAMPBELL.

**ON THE TREATMENT OF CRIMES COMMITTED BY  
(341) ALCOHOLICS.** (Zur Behandlung alkoholischer Delikte.) O.  
JULIUSBURGER (of Steglitz), *Psych.-Neur. Wchnschr.*, April 7,  
1906.

THE author agrees with Hoppe that the only suitable treatment for inebriates who have committed misdemeanours under the influence of drink is to send them, not to prison, but to an inebriate



institution. By punishment one gains nothing. He does not agree with Hoppe's suggestion as to the practical method of treating the first offence, but holds that on the first offence the inebriate should be committed to an institution for treatment. He emphasises his own views as to the importance of the educational and ethical aspects of the treatment of this class of individuals.

C. MACFIE CAMPBELL.

## Bibliography

### ANATOMY

- GEMELLI. Della struttura delle cellule nervose. *Riv. Speriment. di Freniatria*, Vol. xxxii., 1906, p. 212.  
 KOHN. Ganglienzelle und Nervenfasern. *Münch. med. Woch.*, Juli 3, 1906, p. 1306.  
 DONAGGIO. Procedimento supplementare dei metodi alla piridina per la rapida differenziazione del reticolo fibrillare negli elementi nervosi. *Riv. Speriment. di Freniatria*, Vol. xxxii., 1906, p. 394.  
 EISATH. Über normale und pathologische Histologie der menschlichen Neuroglie. *Monatssch. f. Psychiat. u. Neurol.*, Juli, p. 1.  
 QUENSEL. Beiträge zur Kenntnis der Grosshirnfaserung. *Monatssch. f. Psychiat. u. Neurol.*, Juli, p. 36.  
 NAGEOTTE. The Pars Intermedia or Nervus Intermedius of Wrisberg, and the Bulbo-Pontine Gustatory Nucleus in Man. *Rev. Neurol. and Psychiat.*, July 1906, p. 473.  
 REICHARDT. Ueber die Untersuchung des gesunden und kranken Gehirnes mittels der Wage. Fischer, Jena, 1906, M. 2.40.  
 J. T. WILSON. On the Anatomy of the Calamus Region in the Human Bulb; with an account of a hitherto undescribed "nucleus postremus." Part ii. *Journ. of Anat. and Physiol.*, July 1906, p. 357.  
 W. B. CLARK. The Cerebellum of *Petromyzon fluoratilis*. *Journ. of Anat. and Physiol.*, Vol. xl., Part iv., p. 318.  
 DAVID ORR. The Descending Degenerations of the Posterior Columns in (1) Transverse Myelitis, and (2) after Compression of the Dorsal Posterior Roots by Tumours. *Rev. Neurol. and Psychiat.*, July 1906, p. 488.

### PHYSIOLOGY

- RIEGER. Untersuchungen über Muskelzustände. Fischer, Jena, 1906, M. 2.  
 J. FROUDE FLASHMAN. A Preliminary Note on the Motor Areas in the Cerebral Cortex of Marsupials. *Reports from Pathol. Lab. of Lunacy Depart., N.S.W. Government*, Vol. i., Part ii., 1906, p. 24.  
 OHANNESSIAN. Le velocità nella scrittura. *Riv. di Patol. nerv. e ment.*, Vol. xi., f. 5, 1906, p. 207.  
 R. G. HARRISON. Further Experiments on the Development of Peripheral Nerves. *Am. Journal of Anatomy*, Vol. v., No. 2, p. 121.  
 DOGIEL u. ARCHANGELSKY. Der Bewegungshemmende und der motorische Nervenapparat des Herzens. *Arch. f. d. gesamte Physiologie*, Bd. 103, H. 142, 1906, p. 1.

### PSYCHOLOGY

- DEWEY. The Experimental Theory of Knowledge. *Mind*, July 1906, p. 293.  
 J. S. MACKENZIE. The New Realism and the Old Idealism. *Mind*, July 1906, p. 308.

- ABB. Kritik des Kant'schen Apriorismus von Standpunkt des reinen Empirismus aus unter besonderer Berücksichtigung von J. S. Mill und Mach. *Arch. f. d. gesamte Psychologie*, Bd. 7, H. 3 u. 4, p. 227.
- W. M'DOUGALL. Physiological Factors of the Attention-Process (IV.), Conclusion. *Mind*, July 1906, p. 329.
- FOSTER WATSON. The Freedom of the Teacher to Teach Religion. *Mind*, July 1906, p. 360.

## PATHOLOGY

- MEDEA. Contributo allo studio delle fini alterazioni della fibra nervosa, ecc. *Riv. Speriment. di Freniatria*, Vol. xxxii., 1906, p. 325.
- BESTÀ. Sopra la degenerazione e rigenerazione (in seguito al taglio) delle fibre nervose periferiche. *Riv. Speriment. di Freniatria*, Vol. xxxii., 1906, p. 99.
- NAGEOTTE. Régénération collatérale des fibres nerveuses terminées par des masses de croissance, à l'état pathologique et à l'état normal. *Nouv. Icon. de la Salpêtrière*, mai-juin 1906, p. 217.
- RIVA. Lesioni del reticolo neuro-fibrillare della cellula nervosa nell' inanizione sperimentale studiate con i metodi del Donaggio. *Riv. Speriment. di Freniatria*, Vol. xxxii., 1906, p. 400.
- MARINESCO. Lésions fines des Centres Nerveux au cours des Polynévrites. (Soc. de Neurol.) *Rev. Neurol.*, juin 30, 1906, p. 591.
- LEONARD S. DUDGEON. A Study of the Various Changes which occur in the Tissues in Acute Diphtheritic Toxæmia, more especially in Reference to "Acute Cardiac Failure." *Brain*, Vol. xxix., No. 114, 1906, p. 227.
- DONAGGIO. Effetti dell' azione combinata del digiuno e del freddo sui centri nervosi di mammiferi adulti. *Riv. Speriment. di Freniatria*, Vol. xxxii., 1906, p. 373.
- ANDRÉ THOMAS. Les Névromes de régénération dans un cas d'Amputation de la cuisse. (Soc. de Neurol.) *Rev. Neurol.*, juin 30, 1906, p. 575.
- ITALO ROSSI. Malformation du Cervelet. (Soc. de Neurol.) *Rev. Neurol.*, juin 1906, p. 567.
- CENI. Di un caso di amelia sperimentale. *Riv. Speriment. di Freniatria*, Vol. xxxii., 1906, p. 133.
- J. HERBERT PARSONS and GEORGE COATS. A Case of Orbital Encephalocele with Unique Malformations of the Brain and Eye. *Brain*, Vol. xxix., No. 114, 1906, p. 209.
- J. FROUDE FLASHMAN. Internal Features of the Brain of a Microcephalic Idiot, showing lack of Corpus Callosum. *Reports from Pathol. Labor. of Lunacy Depart., N.S.W. Government*, Vol. i., Part ii., 1906, p. 1.
- DENUCE. Spina Bifida. Anatomie pathologique et embryogénie. Doin, Paris 1906, 10 fr.
- WEBER. De quelques altérations du tissu cérébral dues à la présence du tumeurs. *Nouv. Icon. de la Salpêtrière*, mai-juin 1906, p. 247.
- MIRALLIÉ. Note sur l'histologie pathologique de la paroi de l'abcès cérébral. *Arch. de Neurol.*, juin 1906, p. 460.
- CERLETTI. Sull' anatomia patologica della paralisi progressiva. (Rassegna critica.) *Riv. Speriment. di Freniatria*, Vol. xxxii., 1906, p. 410.

## CLINICAL NEUROLOGY AND PSYCHIATRY

## GENERAL—

- W. BATESON. An Address on Mendelian Heredity and its Application to Man. *Brain*, Vol. xxix., No. 114, 1906, p. 157.
- BUSCHAN. Gehirn und Kultur. Bergmann, Wiesbaden, 1906, M. 1.60.
- KOLLARITS. Beiträge zur Kenntnis der vererbten Nervenkrankheiten. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 30, H. 5-6, 1906, S. 293.
- MERZBACHER. Sull' importanza diagnostica della puntura lombare nella psichiatria e neurologia. *Riv. di Patol. nerv. e ment.*, Vol. xi., f. 5, 1906, p. 193.
- BELOUSOW. Delineatio synoptica nervorum hominis. Mit begleit. deutschen Text und eine französische Übersetzung, von Krause und Nicolas. Urban und Schwarzenberg, Wien, 1906, M. 100.
- STIEDA. Ueber die Psychiatrie in Japan. *Centralbl. f. Nervenheilk. u. Psychiat.*, Juli 1, 1906, S. 514.
- J. FROUDE FLASHMAN. Report to the Inspector-General of Insane, New South Wales. *Reports from Pathol. Lab. of Lunacy Depart., N.S.W. Government*, Vol. i., Part ii., 1906, p. 64.

**MUSCLES—**

CATTANEO. Sulle paralisi dei neonati e sulla myatonie generalizzata di Oppenheim. *La Clinica Moderna*, 13 Giugno, p. 283.

**PERIPHERAL NERVES—**

W. K. HUTTON. Remarks on the Innervation of the Dorsum Manus, with special reference to certain rare abnormalities. *Journ. of Anat. and Physiol.*, July 1906, p. 326.

SYDNEY SCOTT. A Record of the Decussations of the Brachial Plexus in Man. *Journ. of Anat. and Physiol.*, July 1906, p. 412.

GROBER. Zur Kasuistik der neuritischen Plexualähmung. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 30, H. 5-6, 1906, S. 424.

HENLE. Über Kriegsverletzungen der peripherischen Nerven. *Arch. f. Klin. Chirurg.*, Bd. 79, H. 4, p. 1070.

BIANCHINI. Le nevriti infettive, etiologia e patologia generale, studio critico e sperimentale. Nicola Zanichelli, Bologna, 1906.

BURR. Alcoholic Multiple Neuritis. *Am. Journ. of the Med. Sci.*, July 1906, p. 77.

W. G. SPILLER and W. T. LONGCOPE. Multiple Motor Neuritis, including Landry's Paralysis and Lead Palsy. *Medical Record*, June 21, 1906, p. 81.

RUYSSEN. De l'exagération des réflexes dans les polynévrites. (*Thèse*.) Le Bigot frères, Lille, 1906.

PIERRE MARIE. Forme spéciale de Névrite interstitielle hypertrophique progressive de l'enfance. (Soc. de Neurol.) *Rev. Neurol.*, juin 30, 1906, p. 557.

**SPINAL CORD—**

**Tabes.**—EULENBERG. Wesen u. Pathologie der Tabes. *Wien. med. Woch.*, Juli 28, p. 1538.

ANDRÉ THOMAS et HAUSER. Tabes avec lésions à peine appréciables de la Moelle. (Soc. de Neurol.) *Rev. Neurol.*, juin 30, 1906, p. 573.

ODDO. Tabes avec Amyotrophie et Arthropathie suppurée. (Soc. de Neurol.) *Rev. Neurol.*, juin 30, 1906, p. 587.

VERGER et GRENIER DE CARDENAL. Tabes pendant l'évolution duquel apparaît un chancre vraisemblablement syphilitique. Retard dans l'évolution anatomique des lésions médullaires. Névrites périphériques intenses en rapport avec une arthropathie du genou. *Rev. Neurol.*, juillet 15, 1906, p. 602.

ACHILLE JOLLY. Crises Hépatiques et Tabes. (*Thèse*.) Henri Jouve, Paris, 1906.

MOUTIER et JEAN DEROIDE. Arthropathie Tabétique de la hanche (type atrophique) et du genou (type hypertrophique). (Soc. de Neurol.) *Rev. Neurol.*, juin 30, 1906, p. 568.

CLAUDE et TOUCHARD. Tabes fruste avec Arthropathie hypertrophique. (Soc. de Neurol.) *Rev. Neurol.*, juin 30, 1906, p. 563.

A. D. YOUNG. The Treatment of Tabes in the Peataxic Stage. *New York Med. Journ.*, July 7, 1906, p. 22.

**Poliomyelitis Anterior Acuta.**—ARMAND-DELILLE et BOUDET. Un cas de Poliomyélite antérieure subaiguë diffuse de la première enfance, avec autopsie. (Soc. de Neurol.) *Rev. Neurol.*, juin 30, 1906, p. 579.

R. T. TAYLOR. Operative Treatment of Infantile Paralysis with especial reference to Neuroplasty. *New York Med. Journ.*, July 7, 1906, p. 9.

VULPIUS. Erfahrungen in der Behandlung der spinalen Kinderlähmung. *Munch. med. Woch.*, Juli 24, 1906, p. 1451.

**Progressive Muscular Atrophy.**—ARCHIBALD CHURCH. The Neuritic Type of Progressive Muscular Atrophy. A Case with Marked Heredity. *Journ. Nerv. and Ment. Dis.*, July 1906, p. 447.

**Amyotrophic Lateral Sclerosis.**—CULLERRE. Troubles mentaux dans la sclérose latérale amyotrophique. *Arch. de Neurol.*, juin 1906, p. 433.

**Paraplegia.**—GAUSSEL. Étude pathogénique de la paraplégie du mal de Pott. *Arch. de méd. expér.*, mai 1906, p. 293.

DEJERINE et CAMUS. Un cas de Mal de Pott cervical avec troubles très étendus de la Sensibilité par Méningite concomitante. (Soc. de Neurol.) *Rev. Neurol.*, juin 30, 1906, p. 560.

CHARLES PETIT et VEILLARD. Paraplégie Spasmodique. Troubles cérébraux. Sclérose en plaques probable. (Soc. de Neurol.) *Rev. Neurol.*, juin 30, 1906, p. 595.

- PELLIZZI.** Paraplegia spasmodica familiare e demenza precoce. *Riv. Speriment. di Freniatria*, Vol. xxxii., 1906, p. 1.
- LEJONNE et LHERMITTE.** Étude sur les paraplégies par rétraction chez les vieillards. *Nouv. Icon. de la Salpêtrière*, mai-juin 1906, p. 255.
- Myelomalacia.**—**RAYMOND et ALQUIER.** Myélomalacie incomplète avec Ostéite raréfiante d'un corps vertébral ayant simulé une Compression subaiguë de la Moelle. (Soc. de Neurol.) *Rev. Neurol.*, juin 30, 1906, p. 581.
- Multiple Sclerosis.**—**E. W. TAYLOR.** Multiple Sclerosis: a Contribution to its Clinical Course and Pathological Anatomy. *Journ. Nerv. and Ment. Dis.*, June 1906, p. 351.
- Syringomyelia.**—**ARCHIBALD CHURCH.** Syringomyelia, with Involvement of Cranial Nerves probably. A Syringobulbia. *Journ. Nerv. and Ment. Dis.*, July 1906, p. 454.
- RAYMOND et LHERMITTE.** Sur un cas de Syringomyélie à type douloureux. (Soc. de Neurol.) *Rev. Neurol.*, juin 30, 1906, p. 576.
- W. B. RANSOM.** A Case of Syringomyelia and Adenoglioma of the Spinal Cord. *Journ. Pathol. and Bacteriol.*, June 1906, p. 364.
- GILBERT BAILLET et MAILLARD.** Syringomyélie à forme anormale? (Soc. de Neurol.) *Rev. Neurol.*, juin 30, 1906, p. 562.
- Conus Lesions.**—**MINOR.** Zur Pathologie des Epiconus medullaris. *Deutsch. Ztschr. f. Nervenheilk.*, Bd. 30, H. 5-6, 1906, S. 389.
- FISCHLER.** Ein Beitrag zur Kenntnis der traumatischen Conus-läsionen. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 30, H. 5-6, 1906, S. 364.
- L. R. MÜLLER.** Über die Extirpation der unteren Hälfte des Rückenmarks und deren Folgeerscheinungen. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 30, H. 5-6, 1906, S. 413.

# **BRAIN—**

- Meningeal Hæmorrhage.**—**APELT.** Zum Kapitel der Diagnose des extra- und intraduralen traumatischen und pachymeningitischen Hämatoma. *Mitteil. aus der Grenzgeb. der Med. and Chir.*, Bd. 16, H. 2.
- Meningitis.**—**PETERS.** Ueber die Entzündung des extraduralen Gewebes des Rückenmarks bei der Genickstarre. *Deutsch. med. Woch.*, Juli 19, 1906, p. 1151.
- WEICHSELBAUM.** Meningitis cerebro-spinalis. *Wien. med. Woch.*, Juli 14, 1906, p. 1451.
- WILLIAM WRIGHT and WILLIAM ARCHIBALD.** Epidemic Cerebro-spinal Meningitis, with Notes on Recent Cases occurring in Glasgow. *Lancet*, June 30, 1906, p. 1815.
- ALBERT CHARPENTIER.** Méningite chronique syphilitique conjugale. (Soc. de Neurol.) *Rev. Neurol.*, juin 30, 1906, p. 550.
- RAYMOND et BAUR.** Syndrome de Ménière dû à une Méningite de la base. (Soc. de Neurol.) *Rev. Neurol.*, juin 30, 1906, p. 584.
- HILDESCHIM.** The Prognosis in Posterior Basic Meningitis. *Pediatrics*, July 1906, p. 431.
- Encephalitis.**—**BREGMAN.** Über eine diffuse Encephalitis der Brücke mit Ausgang in Heilung. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 30, H. 5-6, 1906, S. 450.
- Tumour.**—**THOMAS BUZZARD.** A Clinical Lecture on Two Cases illustrating Points in the Diagnosis of Tumour or other Lesion of the Temporo-Sphenoidal Lobe. *Lancet*, June 30, 1906, p. 1807.
- GEORGE A. MOLEEN.** Subcortical Cerebral Gumma Accurately Localised in the Comatose State: Death; Autopsy. *Journ. Nerv. and Ment. Dis.*, June 1906, p. 407.
- C. T. VAN VALKENBERG.** Tumor in der Marksubstanz der motorischen Zone (Armregion). Zur Differentialdiagnose zwischen kortikalem und subkortikalem Sitz des Herdes. *Neurol. Centralbl.*, Juli 1, 1906, S. 594.
- LIEBSCHER.** Zur Kenntnis der Zystizerkose des Gehirns mit Geistesstörung. *Prag. med. Woch.*, June 23, p. 339.
- J. A. MACDONALD.** Report of a Case of Brain Tumour. *Journ. of Am. Med. Ass.*, July 14, 1906, p. 101.
- GIORDANI.** Sur le diagnostic des Tumeurs de l'Hypophyse par la radiographie. (Thèse.) Baillière et fils, Paris, 1906.
- VOLPI-GHIRARDINI.** Considerazioni sopra un caso di tumore comprimente la metà destra del ponte di varolio diagnosticato in vita. *Riv. Speriment. di Freniatria*, Vol. xxxii., 1906, p. 166.
- Abscess.**—**DUPRÉ et DEVAUX.** Abscess cérébral; nécrose corticale; syndrome méningé. *Nouv. Icon. de la Salpêtrière*, mai-juin 1906, p. 239.

- Hereditary Cerebellar Ataxia.**—RAYMOND et ROSE. Héréd-ataxie cérébelleuse. (Soc. de Neurol.) *Rev. Neurol.*, juin 30, 1906, p. 546.
- Cerebral Diplegia.**—POYNTON, PARSONS and GORDON HOLMES. A Contribution to the Study of Amaurotic Family Idiocy. *Brain*, Vol. xxxix., No. 114, 1906, p. 180.
- General Paralysis.**—GIUNIO CATÒLA. A proposito della patogenesi della paralisi progressiva e dello spirochaeta pallida di Schaudinn-Hauffmann. *Riv. di Patol. nerv. e ment.*, Vol. xi., f. 5, 1906, p. 218.
- ETIENNE et PERRIN. Arthropathie nerveuse chez un paralytique général non tabétique. *Nouv. Icon. de la Salpêtrière*, mai-juin 1906, p. 276.
- ADAM. Ein Fall progressiver Paralyse im Anschluss an einen Unfall durch elektrischen Starkstrom. *Allg. Zeit. f. Psychiat.*, Bd. 63, H. 3 u. 4, p. 428.
- HUGH T. PATRICK. Hereditary Cerebellar Ataxia and General Paresia. *Journ. Nerv. and Ment. Dis.*, July 1906, p. 459.
- Pellagra.**—C. CENI. Sul ciclo biologico dei penicilli verdi in rapporto coll'endemia pellagrosa. *Riv. Speriment. di Freniatria*, Vol. xxxii., 1906, p. 184.

#### MENTAL DISEASES—

- WERNICKE. Grundriss der Psychiatrie in klinischen Vorlesungen. Thieme, Leipzig, 1906, M. 14.
- HELLPACH. Der Gegenstand der Psychopathologie. *Arch. f. d. gesamte Psychologie*, Bd. 7, H. 344, p. 143.
- ROSENFELD. Über den Einfluss psychischer Vorgänge auf den Stoffwechsel. *Allg. Zeit. f. Psychiat.*, Bd. 63, H. 3 u. 4, p. 367.
- ALBRECHT. Die psychischen Ursachen der Melancholie. *Monatssch. f. Psychiat. u. Neurol.*, Juli, p. 65.
- ALBRECHT. Manisch-depressiver Irresein und Arteriosklerose. *Allg. Zeit. f. Psychiat.*, Bd. 63, H. 3 u. 4, p. 402.
- LUGIATO e OHANNESSIAN. La pressione sanguigna nei malati di mente. *Riv. Speriment. di Freniatria*, Vol. xxxii., 1906, p. 225.
- MARGULIES. Über graphisch-kinästhetische Halluzinationen. *Neurol. Centralbl.*, Juli 16, 1906, S. 651.
- GROSZMANN. The Position of the Atypical Child. *Journ. Nerv. and Ment. Dis.*, July 1906, p. 425.
- BOURNEVILLE et BORD. Types d'idiotie. Cas d'idiotie mongolienne. *Rev. d'hygiène de médecine infantiles*, T. v., No 3, p. 221.
- PIGHINI. Il ricambio organico nella demenza precoce. *Riv. Speriment. di Freniatria*, Vol. xxxii., 1906, p. 355.
- DE-SANCTIS. Sopra alcune varietà della demenza precoce. *Riv. Speriment. di Freniatria*, Vol. xxxii., 1906, p. 141.
- D'ORMEA. Il potere riduttore delle urine nei dementi precoci. *Riv. Speriment. di Freniatria*, Vol. xxxii., 1906, p. 79.
- ORBISON. Acute Paranoia exhibiting cyclical Relapses. *Am. Journ. of the Med. Sci.*, July 1906, p. 91.
- GREGOR. Ein Beitrag zur Kenntnis des intermittierenden Irreseins. *Allg. Zeit. f. Psychiat.*, Bd. 63, H. 3 u. 4, p. 417.
- GEIST. Über kombinierte Psychosen nebst einem kasuistischen Beitrag. *Allg. Zeit. f. Psychiat.*, Bd. 63, H. 3 u. 4, p. 434.
- RAW. Mental Disorders of Pregnancy and the Puerperal Period. *Edin. Med. Journ.*, Aug. 1906, p. 118.
- A. MARIE. La Démence. Doin, Paris, 1906, 4 fr.
- J. MOREL. Prophylaxie et traitement des criminels récidivistes. *Journ. de Neurol.*, juin 20, 1906, p. 221.

#### ALCOHOL—

- HARRY CAMPBELL. The Alcoholic Craving. *Brit. Journ. Inebriety*. July 1906, p. 5.
- FORLÌ. Sulla polineurite reumatica dei nervi cranici. *Riv. Speriment. di Freniatria*, Vol. xxxii., 1906, p. 280.
- JEAN GALEZOWSKI. Les manifestations oculaires du saturnisme. *Arch. gén. de méd.*, juin 26, 1906, p. 1613.
- V. HIPPEL. Ueber seltene Fälle von Lähmung der Akkommodation und von Pupillenstarre. *Klin. Monatsblatt. f. Augenheilk.*, Juli 1906, p. 97.
- TÖDTER. Ein Beitrag zur isolierten Blicklähmung nach oben und unten. *Klin. Monatsblatt. f. Augenheilk.*, Juli 1906, p. 102.
- HAMMERSCHLAG. Behandlung der Trigeminusneuralgie mit Perosmium Säure. *Arch. f. klin. Chirurg.*, Bd. 79, H. 4, p. 1050.

- FERNAND LÉVY. Essai sur les névralgies faciales. Rousset, Paris, 1906, 5 fr.  
 JACOBSON. Ueber traumatisch-chirurgische Facialislähmungen. *Deutsch. med. Woch.*, Juli 19, 1906, p. 1153.  
 ALLAIRE. Sur deux cas de paralysie faciale protubérantielle. *Ann. d'Électrobiol. et de Radiol.*, avril 1906, p. 227.  
 MINGAZZINI und ASCENZI. Klinischer Beitrag zum Studium der Hemiatrophie der Zunge supranuklearen Ursprungs. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 30, H. 5-6, 1906, S. 437.

## GENERAL AND FUNCTIONAL DISEASES—

- Epilepsy.**—BUSCHAN. Die Epilepsie. Konegan, Leipzig, 1906, M. 1.40.  
 Sir WILLIAM GOWERS. Clinical Lectures on the Borderland of Epilepsy: Vertigo. *Brit. Med. Journ.*, July 21, 1906, p. 128.  
 MORSELLI e PASTORE. Le modificazioni qualitative e quantitative delle cellule eosinofile nel sangue degli epilettici. *Riv. Speriment. di Freniatria*, Vol. xxxii., 1906, p. 258.  
 A. E. RUSSELL. Cessation of the Pulse during the Onset of Epileptic Fits, with Remarks on the Mechanism of Fits. *Lancet*, July 21, 1906, p. 152.  
 BESTA. Ricerche sopra la pressione sanguigna. Il polso e la temperatura degli epilettici. *Riv. Speriment. di Freniatria*, Vol. xxxii., 1906, p. 306.  
 RICCI. Studio critico sopra 393 casi di epilessia. *Riv. Speriment. di Freniatria*, Vol. xxxii., 1906, p. 291.  
 FRIEDMANN. Über die nicht epileptischen Absenzen oder kurzen narkoleptischen Anfälle. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 30, H. 5-6, 1906, S. 462.  
**Neurasthenia.**—HUCHARD. The Mental State of Neurasthenic Patients. *Paris Med. Journ.*, July 1906, p. 91.  
 CRAMER. Die Nervosität, ihre Ursachen, Erscheinungen und Behandlung. Fischer, Jena, 1906, M. 8.  
 NEVILLE HART. The Nervous Phenomena following Attempted Suicide by Hanging. *Lancet*, June 30, 1906, p. 1821.  
**Traumatic Neurasthenia.**—WILLY HELLPACH. Unfallsneurosen und Arbeitsfreude. *Neurol. Centralbl.*, Juli 1, 1906, S. 605.  
**Hysteria.**—DEBOVE. Dysarthrie hystérique. *Arch. gén. de méd.*, juillet 10, 1906, p. 1746.  
 HENRI CLAUDE. Troubles Vasomoteurs de nature Hystérique. (Soc. de Neurol.) *Rev. Neurol.*, juin 30, 1906, p. 551.  
 RAYMOND et LEJONNE. Astasie-Abasie fonctionnelle avec association de phénomènes organiques. (Soc. de Neurol.) *Rev. Neurol.*, juin 1906, p. 564.  
 FISCHL. Les vomissements acétonémiques et l'hystérie infantile. *Rev. mens. des maladies de l'enfance*, juillet 1906, p. 289.  
 CHARPENTIER. Dégénérescence mentale et hystérie. (Thèse.) Durand, Paris, 1906.  
 SPECHT. Ueber Hysteromelancholie. *Centralbl. f. Nervenheilk. u. Psychiat.*, Juli 13, 1906, S. 545.  
 SADGER. Die Hydriatik der Hysterie und der Zwangneurose. *Centralbl. f. Nervenheilk. u. Psychiat.*, Juli 1, 1906, S. 505.  
**The Tics.**—ROUBINOVITCH. Iconographie de l'évolution d'un cas de maladie des tics. *Nouv. Icon. de la Salpêtrière*, mai-juin 1906, p. 289.  
**Tetany.**—Frankl-Hochwart. Die Prognose der Tetanie der Erwachsenen. *Neurol. Centralbl.*, Juli 16, 1906, S. 642.  
**Exophthalmic Goitre.**—BERNHARDT. Zur Pathologie der Basedow'schen Krankheit. *Berlin. klin. Woch.*, Juli 2, 1906, p. 905.  
 SCHULTZE. Zur Chirurgie des Morbus Basedow. *Mitteil. aus der Grenzgeb. der Med. und Chir.*, Bd. 16, H. 2, 1906.  
 SAINTON. Les traitements actuels du goitre exophthalmique. Levé, Paris, 1906.

## MISCELLANEOUS SYMPTOMS—

- LAMBERT LACK. Headache of Nasal Origin. *Practitioner*, July 1906, p. 46.  
 WALTER H. JESSOP. Eye-Strain as a Cause of Headache. *Practitioner*, July 1906, p. 40.  
 WILFRED HARRIS. Migraine and Toxæmic Headaches. *Practitioner*, July 1906, p. 25.  
 JAMES TAYLOR. Intracranial Disease as a Cause of Headache. *Practitioner*, July 1906, p. 21.  
 ROBERT SAUNDBY. Headache of Renal Origin. *Practitioner*, July 1906, p. 19.

- CAMPBELL THOMSON. The Causation and Treatment of Headaches. Introduction. *Practitioner*, July 1906, p. 15.
- LÉVI. Migraine thyroïdienne. *Rev. d'Hygiène et de Médecine infantiles*, T. v., No. 3, 1906, p. 246.
- SPIELMEYER. Hemiplegie bei intakter Pyramidenbahn. *Münch. med. Woch.*, Juli 17, 1906, p. 1404.
- CHOTZEN. Einseitige Temperatursteigerung in der gelähmten Körperhälfte bei zerebraler Herderkrankung. *Münch. med. Woch.*, Juli 3, 1906, p. 1304.
- MAUDSLEY. Brachioptegia of Cerebellar Type and Rhythmical Tremor. *Inter-Colonial Medical Journ. of Australasia*, June 20, 1906, p. 302.
- NOICA et AVRAMESCU. Sur deux cas de perte du Sens Stéréognostique à topographie nerveuse. (Soc. de Neurol.) *Rev. Neurol.*, juin 30, 1906, p. 592.
- LEJONNE et EGGER. Hémianesthésie d'origine Corticale probable. (Soc. de Neurol.) *Rev. Neurol.*, juin 30, 1906, p. 571.
- JAMES MACKENZIE. Remarks on the Meaning and Mechanism of Visceral Pain as shown by the Study of Visceral and other Sympathetic (Autonomic) Reflexes. *Brit. Med. Journ.*, June 30, 1906, p. 1523.
- ALFRED GORDON. A further Contribution to the Study of the "Paradoxic Reflex." *Journ. Nerv. and Ment. Dis.*, June 1906, p. 415.
- KLIPPEL et MAILLARD. Un cas de maladie de Recklinghausen avec dystrophie multiples et prédominance unilatérale. *Nouv. Icon. de la Salpêtrière*, mai-juin 1906, p. 282.
- PAUL TOUCHARD. Recherches anatomo-cliniques sur la Sclérodémie généralisée. (Thèse.) Steinheil, Paris, 1906.
- Aphasia.**—DEJERINE. L'Aphasie motrice. *La Presse médicale*, 18 juillet 1906, p. 453.
- DEJERINE. L'Aphasie Sensorielle. *Presse méd.*, juillet 1906, p. 437.
- KRAEPELIN. Über Sprachstörungen im Traume. Engelmann, Leipzig, 1906, M. 3.
- BOULENGER. Quelques considérations sur l'écriture en miroir. Les troubles de l'orientation et son éducation. *Journ. de Neurol.*, juillet 5, 1906, p. 241.
- DEJERINE. Considérations sur la soi-disant "aphasie tactile." *Rev. Neurol.*, juillet 15, 1906, p. 597.
- HASKOVEC. Cas particulier d'Infantilisme du Langage chez une femme de 58 ans, survenu après une attaque d'Aphasie motrice. (Soc. de Neurol.) *Rev. Neurol.*, juin 30, 1906, p. 593.
- DEJERINE. Considérations sur la soi-disant "aphasie tactile." (Soc. de Neurol.) *Rev. Neurol.*, juin 30, 1906, p. 553.
- Cerebral Localisation.**—BONHOEFFER. Ueber die Bedeutung der Jackson'schen Epilepsie für die topische Hirndiagnostik. *Berlin. klin. Woch.*, Juli 9, 1906, p. 935.
- ANTON. Symptome der Stirnhirnerkrankung. *Münch. med. Woch.*, Juli 3, p. 1289.
- DEJERINE et ROUSSY. Le Syndrome thalamique. *Rev. Neurol.*, juin 30, 1906, p. 521.

**TREATMENT—**

- SIR WILLIAM BROADBENT. The Treatment of Sleeplessness. *Practitioner*, July 1906, p. 1.
- PAUL PETIT. Zona survenu à l'occasion d'une séance de H. F. et guéri par le courant continu. *Ann. d'Électrobiol. et de Radiol.*, avril 1906, p. 232.
- NEUMANN. Die Heilung der Nervosität durch intelligente Lebenshaltung. Borggold, Leipzig, 1906, M. 1.20.
- IDE. Die Behandlung der Neurasthenie durch das Seeklima. *Neurol. Centralbl.*, Juli 16, 1906, S. 654.
- BABINSKI et DELHERM. Sur le traitement de la Névralgie Faciale par les Courants Voltaïques à intensité élevée. (Soc. de Neurol.) *Rev. Neurol.*, juin 30, 1906, p. 544.
- DETERMANN. Physikalische Therapie der Erkrankungen des Zentralnervensystems inklusive der allgemeinen Neurosen. Enke, Stuttgart, 1906, M. 3.60.

# Review of Neurology and Psychiatry

---

## Original Articles

### ON THE FREQUENCY WITH WHICH CERTAIN SIGNS AND SYMPTOMS OCCUR IN CASES OF DIS- SEMINATED SCLEROSIS BEFORE THE DEVELOP- MENT OF SO-CALLED CARDINAL SIGNS.

By ASHLEY W. MACKINTOSH, M.A., M.D., Aberdeen.

SINCE Charcot emphasised the existence and importance of *formes frustes*, or atypical forms of disseminated sclerosis, it has been recognised that this disease may present for a long period, it may be many years, none of the so-called cardinal signs, but simply the features of spastic paraplegia. I have notes of 110 cases of disseminated sclerosis, including the series of 80 cases on which a former paper in this Review (Vol. i. p. 73) was based. In 30 cases, which eventually appeared clear examples of disseminated sclerosis, the cardinal signs were, when the cases were seen at an earlier stage, either entirely absent, or so doubtful that it was believed that no diagnostic significance could be attached to them; at this early stage all appeared to be examples of simple spastic paraplegia, but it was possible in all to elicit, either from the histories of the cases or from the "present condition," certain signs or symptoms not belonging to the symptomatology proper of primary spastic paraplegia. It is believed that an analysis of the nature and frequency of the most important of these signs and symptoms may be of interest and also of some value from the point of view of the early diagnosis of disseminated sclerosis.



## POINTS IN THE HISTORIES OF THE CASES.

## I. Marked variability or remission of symptoms, 30 cases :—

## (α) Leg-paralysis, 25 cases :—

Paraplegia . . .	19 cases.
Monoplegia . . .	5 „
Hemiparesis . . .	2 „
Unsteady gait . . .	3 „

(β) Paræsthesia of legs, especially of feet, 9 cases  
(variable objective sensory defect in 2) :—

Pains in legs . . . . .	3 cases.
Attacks of numbness of one half of body . . .	1 case.
Markedly severe and remittent pain in spine . .	1 „

## (γ) Sphincter troubles, 7 cases.

## (δ) Arm-affection :—

Paræsthesiæ, especially about fingers . . .	11 cases.
Arms weak or shaky . . . . .	10 „

## (ε) Amblyopia . . . 6 cases.

Diplopia . . . . .	6 „
Squint . . . . .	2 „
Vertigo . . . . .	3 „
Speech defect . . . . .	3 „ (temporarily “difficult,” “indistinct”).

Epileptiform attacks 1 case.

II. Paræsthesiæ, 23 cases (“numbness,” “pins and needles,”  
“hot or cold feeling,” etc.) :—

Arms, chiefly about fingers . . .	18 cases.
Legs „ „ feet . . .	17 „
Marked cord sensation . . . . .	4 „
Numbness of trunk . . . . .	2 „
Numbness of half of body . . . . .	1 case.

Pains in limbs, 7 cases (legs 6, arms 3).

Pains in spine, 6 cases (chiefly in lumbo-dorsal region).

## III. Arm affection : “weak,” “shaky,” “tremulous,” 12 cases.

## IV. Sphincter trouble (apart from constipation), 12 cases:

either retention of urine or, more often, a tendency to incontinence of urine: in 6 cases, defective control over bowel.

V. Amblyopia	.	.	14 cases.
Diplopia	.	.	7 "
Squint	.	.	2 "
Vertigo	.	.	6 "
Speech defect	.	.	3 "
Epileptiform attacks	.	.	2 "

*Remarks.*—That marked variability or remission of symptoms—emphasised by Oppenheim, Uhthoff, Freund, Buzzard, and others—is one of the most striking and most constant characteristics of disseminated sclerosis is amply borne out by these cases. In several, paraplegic attacks occurred again and again, with apparently complete recovery in the intervals; in a few cases the paraplegia was accompanied by great or even complete loss of power over bladder and bowel, which also entirely disappeared. In most of the cases with sphincter troubles, especially those in which the troubles were transitory, there was simply either retention of urine or precipitate micturition or slight definite incontinence of urine.

There are several interesting histories of temporary amblyopia; thus, in one case, sudden complete blindness is stated to have occurred, first of one eye and then of the other eye two years later, lasting several months and then completely disappearing. In about one-half of the cases of amblyopia, the condition was mainly or entirely unilateral.

Attacks of vertigo were the specially prominent early symptom in one case. They came on quite suddenly and made the patient stagger so that he was "often said to be drunk."

The frequency of occurrence of subjective sensory disturbances (paræsthesiæ, pains) in the early histories of the cases may be noted. Probably these disturbances are rarely absent in cases of disseminated sclerosis, although no note may be made of them. They occur in 93 of my series of 110 cases: paræsthesiæ in 75 cases, pains in 64. The paræsthesiæ generally implicated mainly or solely the distal parts of the limbs (legs 63, arms 53 cases); they were often strikingly transient and prone to recur, and they usually occurred early in the history of a case; in a fair number of cases they were the first symptom noted. The pains affected

the limbs in 39 cases (legs 35, arms 17). Spinal pain, generally in the lumbar or lumbo-sacral region, was noted in 26 cases; in some the pain was very persistent, and not unfrequently it was severe; in one of the present series of 30 cases, severe recurrent attacks of pain in the lower lumbar spine, later tending to shoot down the legs, were the first and most prominent symptom.

POINTS IN THE "PRESENT CONDITION" OF THE CASES, BEFORE  
CARDINAL SYMPTOMS APPEARED.

I. Gait, unsteady or staggering or ataxic, as well as spastic, 18 cases.

II. Primary optic atrophy, 16 cases, ? 2, not noted 2, absent 10.

III. Objective sensory defect of legs, specially feet, 14 cases.

Slight defect of sensation of half of body, 1 case.

Stereognostic sense impaired or lost in one hand, 2 cases.

Sensory defect, chiefly algesic, at tips of fingers, 2 cases.

IV. Paræsthesiæ, 14 cases.

Legs 13, arms 11, trunk 1, girdle sensation 5, pain in lower spine, 7 cases.

V. Sphincter troubles, 9 cases, chiefly tendency to incontinence of urine or precipitate micturition, incontinence of fæces in 2 cases.

VI. Diminution or loss of abdominal and epigastric reflexes, 12 cases.

The state of these reflexes is noted in 15 cases only.

VII. Ocular signs: Pupils unequal, 2 cases.

One pupil sluggish to light, 1 case.

Paresis of external rectus, 3 cases.

Paresis of internal rectus, 2 cases.

*Remarks.*—In 18 cases (*i.e.* 60 per cent.), an ataxic or unsteady element in the gait was noted; in several cases the degree of unsteadiness was very suggestive of cerebellar trouble; in 4 cases it was the only objective sign, apart from spastic paraplegia.

Optic atrophy was present in 16 cases, *i.e.* 53·3 per cent.;

mainly or entirely confined to the outer part of disc in 7 cases, and mainly or entirely unilateral in 4 cases. As a rule the degree of pallor of disc was not great, but marked atrophy was present in 2 cases, in which visual acuity was reduced to  $\frac{6}{15}$  and  $\frac{10}{200}$  respectively in the more atrophic eye. In most cases the visual defect was comparatively slight, although only one case with optic atrophy is noted to have had  $V = \frac{6}{6}$ . The fields of vision are recorded in only a few cases; the fields were narrowed in four (all had optic atrophy), central scotoma was present in one (discs pale, especially at outer parts), absent in 5 cases (2 had optic atrophy). Of the 14 cases with a history of amblyopia, 9 were found to have optic atrophy, 2 ? atrophy, 3 normal discs. In the case already mentioned of sudden complete temporary amblyopia, first of one eye and then of the other, the discs were found to be normal. In 7 cases with optic atrophy there was no history of failing vision. In my total series of 110 cases, optic atrophy was noted in 52, i.e. 47.27 per cent. Buzzard found it in 43 per cent., Uhthoff in 52 per cent., Byrom Bramwell in 33.5 per cent. The slightly greater frequency of optic atrophy in my cases seen at an early stage (53.3 per cent.) compared with that found in the total series of 110 cases (47.27 per cent.) is probably a mere coincidence. It may, however, suggest the relatively early incidence of this condition in disseminated sclerosis.

External ocular paresis occurred in 5 cases, and there was a history of transient squint in other two cases.

Objective sensory defect, generally patchy and not amounting to complete loss, was noted on the legs, especially on the feet, in 14 cases. In the great majority of cases the loss of pain sense predominated; it was noted in 12 cases (sense of touch affected in 5, sense of temperature in 5, sense of passive movement in 4 cases). In the total series of 110 cases, objective sensory defect was noted in 47 (i.e. 42.7 per cent.), the legs being affected in 44, and arms, often very slightly, in 18.

The epigastric and abdominal reflexes were absent in 8, sluggish in 4, normal in 1, and active in 2 cases; thus 12 of 15 cases, seen at an early stage, showed absence or diminution of these reflexes. This gives some support to Müller's contention that the loss of the abdominal reflexes is of diagnostic value.

Sphincter troubles and paræsthesiæ have already been re-

ferred to. The record of paræsthesiæ at the date of examination is incomplete. Among 12 of the most recent and most carefully recorded cases, paræsthesiæ are noted as present in 11.

The four objective signs specially noted, in addition to spastic paraplegia, were optic atrophy, sensory defect, unsteady gait, sphincter troubles. Analysing the 30 cases from the point of view of the frequency with which one or more of these signs occurred, we find :—

Spastic paraplegia alone, 1 case.

Spastic paraplegia and one sign, 11 cases (optic atrophy 2, unsteady gait 4, sensory loss 5).

Spastic paraplegia and two signs, 8.

Spastic paraplegia and three signs, 7.

Spastic paraplegia and all four signs, 3.

The most frequent combinations of two signs were : optic atrophy and unsteady gait (10 cases), sensory defect and unsteady gait (10 cases), optic atrophy and sensory defect (9 cases, in 2 other cases ? optic atrophy).

A certain diagnosis of disseminated sclerosis, especially in the earlier stages, is often difficult or even impossible. One may doubt whether the present position of our knowledge warrants the statements of a recent writer (E. W. Taylor, *Journ. Nerv. and Ment. Dis.*, June 1906) that, since Charcot's time, "we have learned that dependence on scanning speech, nystagmus, and so-called intention tremor is fallacious in the extreme," and that Sach's opinion that the diagnosis of multiple sclerosis should be made only if the cardinal symptoms are present is "evidently an erroneous standpoint." All will, however, agree with Taylor's final words on diagnosis : "we should continue to lay stress upon so-called cardinal symptoms, but more upon others almost equally important but far less conspicuous." The nature and relative frequency of some of these subordinate signs in a series of early cases are shown in the analysis given above. Even in the absence of all the cardinal symptoms, the diagnosis of disseminated sclerosis should be entertained as probable, perhaps the most probable, in a case of spastic paraplegia which exhibits one or more of these subordinate signs—notably primary optic atrophy, unsteady gait, sensory defect—and which gives a history of certain symptoms (paræsthesiæ, sphincter trouble, amblyopia,



*Points in the "Present Condition" of the Cases.*

No.	Sex.	Age at Onset.	Interval from Onset.	Transient, Remittent, or Recurrent Symptomata.						Paresthesias.	Pains.	Sphincter Troubles.	Amblyopia.	Diplopia.	Vertigo.
(1)	F	20	12	p	sph	d	sq	a	sp	vert					
(2)	F	32	12	p	sph	d	sq	a	sp	vert					
(3)	F	42	1½	p	sph	d	sq	a	sp	vert					
(4)	F	19	6	p	sph	d	sq	a	sp	vert					
(5)	F	23	18	p	sph	d	sq	a	sp	vert					
(6)	F	42	6	p	sph	d	sq	a	sp	vert					
(7)	M	27	30	p	sph	d	sq	a	sp	vert					
(8)	F	23	4	p	sph	d	sq	a	sp	vert					
(9)	F	25	12	p	sph	d	sq	a	sp	vert					
(10)	M	35	4	p	sph	d	sq	a	sp	vert					
(11)	F	27	1½	p	sph	d	sq	a	sp	vert					
(12)	M	26	2	p	sph	d	sq	a	sp	vert					
(13)	F	37	5	p	sph	d	sq	a	sp	vert					
(14)	M	37	8	p	sph	d	sq	a	sp	vert					
(15)	F	25	8	p	sph	d	sq	a	sp	vert					
(16)	F	32	3	p	sph	d	sq	a	sp	vert					
(17)	M	43	7	p	sph	d	sq	a	sp	vert					
(18)	M	15	1½	p	sph	d	sq	a	sp	vert					
(19)	F	19	20	p	sph	d	sq	a	sp	vert					
(20)	M	31½	6	p	sph	d	sq	a	sp	vert					
(21)	F	80	3	p	sph	d	sq	a	sp	vert					
(22)	F	23½	1½	p	sph	d	sq	a	sp	vert					
(23)	F	19	30	p	sph	d	sq	a	sp	vert					
(24)	M	31	7	p	sph	d	sq	a	sp	vert					
(25)	M	31	8	p	sph	d	sq	a	sp	vert					
(26)	M	32	10	p	sph	d	sq	a	sp	vert					
(27)	F	26	5	p	sph	d	sq	a	sp	vert					
(28)	F	19	4	p	sph	d	sq	a	sp	vert					
(29)	F	16	1	p	sph	d	sq	a	sp	vert					
(30)	F	19½	4½	p	sph	d	sq	a	sp	vert					

p = motor paralysis.  
s = sensory disturbances (paresthesias or pains).

sph = sphincter troubles.  
d = diplopia.

sq = squint.  
a = amblyopia.

sp = speech defect.  
vert = vertigo.

*Points in the History of the Cases.*

No.	Sex.	Age at Onset.	Interval from Onset.	Transient, Remittent, or Recurrent Symptomata.						Paresthesias.	Pains.	Sphincter Troubles.	Amblyopia.	Diplopia.	Vertigo.
(1)	F	20	12	p	sph	d	sq	a	sp	vert					
(2)	F	32	12	p	sph	d	sq	a	sp	vert					
(3)	F	42	1½	p	sph	d	sq	a	sp	vert					
(4)	F	19	6	p	sph	d	sq	a	sp	vert					
(5)	F	23	18	p	sph	d	sq	a	sp	vert					
(6)	F	42	6	p	sph	d	sq	a	sp	vert					
(7)	M	27	30	p	sph	d	sq	a	sp	vert					
(8)	F	23	4	p	sph	d	sq	a	sp	vert					
(9)	F	25	12	p	sph	d	sq	a	sp	vert					
(10)	M	35	4	p	sph	d	sq	a	sp	vert					
(11)	F	27	1½	p	sph	d	sq	a	sp	vert					
(12)	M	26	2	p	sph	d	sq	a	sp	vert					
(13)	F	37	5	p	sph	d	sq	a	sp	vert					
(14)	M	37	8	p	sph	d	sq	a	sp	vert					
(15)	F	25	8	p	sph	d	sq	a	sp	vert					
(16)	F	32	3	p	sph	d	sq	a	sp	vert					
(17)	M	43	7	p	sph	d	sq	a	sp	vert					
(18)	M	15	1½	p	sph	d	sq	a	sp	vert					
(19)	F	19	20	p	sph	d	sq	a	sp	vert					
(20)	M	31½	6	p	sph	d	sq	a	sp	vert					
(21)	F	80	3	p	sph	d	sq	a	sp	vert					
(22)	F	23½	1½	p	sph	d	sq	a	sp	vert					
(23)	F	19	30	p	sph	d	sq	a	sp	vert					
(24)	M	31	7	p	sph	d	sq	a	sp	vert					
(25)	M	31	8	p	sph	d	sq	a	sp	vert					
(26)	M	32	10	p	sph	d	sq	a	sp	vert					
(27)	F	26	5	p	sph	d	sq	a	sp	vert					
(28)	F	19	4	p	sph	d	sq	a	sp	vert					
(29)	F	16	1	p	sph	d	sq	a	sp	vert					
(30)	F	19½	4½	p	sph	d	sq	a	sp	vert					

diplopia, vertigo), and of variability or remission of one or more symptoms.

The chief details of the individual cases are given in the accompanying table.

*Remarks.*—It will be noted that of the 30 cases, 20 were females and 10 males; in the total series of 110 cases, however, 59 were males and 51 females.

The *age at onset* and *interval from onset* being determined from the histories, *i.e.* from the statements of patients, must be uncertain in many cases, and may be estimated differently by different observers. As stated in the table, the onset was between the ages of 19 and 35 (inclusive) in 24 cases—16 cases between 19 and 27, 8 cases between 30 and 35—below 19 years in 2 cases (15 and 16 years), over 35 years in 4 cases (37, 42, 42, and 43 years).

Some of the cases, in which the interval from onset is stated to have been many years, call for special note, in view of possible error on my part in interpretation of the early symptoms.

Case 1. Twelve years before date of examination, gradual left-sided hemiparesis occurred, with complete recovery; "shortly after," diplopia and squint—also quite temporary; one year later, gradual right-sided hemiparesis appeared, which also was transitory; six years before examination, legs got gradually weak and stiff and varied greatly, but never completely recovered.

Case 2. Twelve years before examination, gradual paraplegia occurred, which disappeared entirely and was diagnosed as functional; there was no other symptom until  $1\frac{1}{2}$  years before examination, when the legs got gradually weak and stiff again and did not recover.

Case 5. Eighteen years before examination there was partial loss of sight, which completely disappeared; 3 years later, temporary paræsthesiæ; no other symptom until 8 years before examination, when legs became weak and numb.

Case 7. During 30 years before examination there were six or eight attacks of great weakness, stiffness and numbness of legs and of arms to less extent; no attack lasted longer than a month; there was complete recovery in intervals until 6 years before examination.

Case 10. Twelve years before examination there was gradual weakness, stiffness, etc., of legs, with incontinence of urine;



patient was bedridden for a year; then gradual complete recovery occurred; later, two slighter attacks of the same character.

Case 19. Twenty years before examination, temporary right-sided hemiparesis occurred, lasting 10 weeks and followed by temporary attacks of weakness of right leg; 4 years later, similar right-sided hemiparesis with partial loss of vision—also quite transient; no other symptom till five years before examination, when gradual weakness, etc., of legs began and did not disappear.

Case 23. Thirty years before examination, sudden temporary numbness of one half of body occurred, and many similar attacks followed; 19 years before examination, weakness and numbness of legs began and never entirely disappeared.

When all due allowance is made for error in estimating the exact date of onset of such cases, the duration of symptoms in some of these, taken in conjunction with evidence of later progression of the disease—as shown *inter alia* by the appearance of cardinal signs—must make one hesitate to speak of recovery from disseminated sclerosis.

---

### PRECOCIOUS PARALYSIS OF THE PALATE IN DIPHTHERIA.

By J. D. ROLLESTON, M.A., M.D. Oxon.,

Assistant Medical Officer at the Grove Fever Hospital of the Metropolitan  
Asylums Board.

SINCE first diphtheritic paralysis was systematically studied, the occurrence of palatal palsy at an early stage of the disease has been well known. Trousseau and Lasègue, and many subsequent writers of the pre-antitoxin era, such as Sanné, Squire, Morell Mackenzie, and Henoch have recorded illustrative cases. Until within recent years, however, no mention is to be found of the significance of the early affection. Some writers, indeed, such as Ruault, Sevestre, and Martin regard the paralyses that develop early as the benign forms, in that they show a tendency to be localised, unlike the late forms which tend to become generalised. An exactly opposite view is held by Baginsky and Romberg in Germany, and in France by Variot, Marfan, Deguy, Berthelot,

Petit, and Babonneix. These authorities hold that precocious paralysis of the palate is a mark of malignancy. The present writer has recently reported some cases that illustrate the truth of this view. The following remarks are based on observations made on 1000 consecutive cases of diphtheria that have been under the present writer's care at the Grove Fever Hospital during the course of the last four years.

Paralysis of the palate occurred in 162 cases (16·2 per cent.). The average date of occurrence was towards the end of the third week (18·3 day among 162 cases).

The term "early" or "precocious" has therefore been applied to such palsies as developed before the beginning of the third week. Fifty such cases among the 162 were so termed, the dates at which they were first observed being as follows:—

*Table I. Showing Date of Occurrence of Precocious Palatal Palsy.*

5th day of disease,	2 cases.
6th   "       "	3   "
7th   "       "	6   "
8th   "       "	7   "
9th   "       "	7   "
10th  "       "	5   "
11th  "       "	7   "
12th  "       "	3   "
13th  "       "	6   "
14th  "       "	4   "

The relation of the onset of the paralysis to the subsidence of acute symptoms is shown in the following table:—

*Table II.*

	Cases.
Palatal palsy noted 3 days before the throat became clean,	4
"       "       2       "       "       "	3
"       "       1       "       "       "	5
"       "       same day as       "       "	10
Death before disappearance of membrane,	3
Palatal palsy noted after throat became clean,	25

Thus in 12 cases the palatal palsy developed before the throat became clean, in 10 the two events took place on the same day, in 3 death occurred before the membrane disappeared,

and in the remaining 25 the palsy arose after the throat had become clean. Most of the cases presented after the membrane had left the throat an extensive superficial necrosis of the epithelium of the tonsils, palate, and uvula, manifested by an opaque appearance of the mucous membrane which sometimes took three weeks or more to be completely regenerated. As will be seen from the following figures, the incidence of precocious palatal palsy is higher in childhood than in adult life:—

*Table III.*

Ages.	Cases of Precocious Palatal Palsy.	Percentage.
0- 5 years	17	4·5
5-10 „	27	5·9
10-15 „	4	4·4
15-20 „	1	3·07
20-30 „	1	

This corresponds with the incidence of post-diphtheritic paralysis at the various ages, as is shown by Table IV.

*Table IV. Ages of 238 Paralysis Cases among 1000 Cases of Diphtheria.*

Ages.	Cases.	Percentage.
0- 5 years	99	26·5
5-10 „	119	26·2
10-15 „	16	17·9
15-50 „	4	4·7

No case of precocious palatal palsy was met with above the age of twenty-nine, though sixteen of the 1000 patients were above that age, 7 of whom had severe, 4 moderate, and 5 mild faucial attacks.

The two sexes were almost equally affected, 22 being males (4·6 per cent.) and 28 females (5·3 per cent.). How frequent an associate early palatal palsy was of the severe forms is illustrated by the fact that of the 50 early cases 20 died, while among the remaining 112 cases of non-precocious palatal palsy there was only one death, due to diaphragmatic paralysis, on the 52nd day.

The mortality of the total 1000 cases was only 78, or 7·8 per cent.

The relation of the frequency of precocious palatal paralysis to the character of the initial faucial attack is shown in the following table :—

*Table V. Showing relation of Precocious Palatal Paralysis to Character of Initial Attack.*

Very severe faucial	.	.	29 cases = 29·2%
Severe faucial	.	.	17 cases = 8·8%
Moderately severe faucial	.	.	1 case = 1·1%
Moderate faucial	.	.	1 case = 0·3%

From this it will be seen that though more frequently found in the severest cases, precocious palatal palsy occasionally follows an angina of only moderate intensity. Berthelot's experience was similar. Four of his twelve cases were of moderate intensity, the remaining eight were severe forms. Petit and Deguy, on the other hand, regard precocious palatal palsy as the exclusive appanage of severe forms. The severity of the attacks might be attributed in the majority of cases to neglect of antitoxin treatment during the first few days of the disease; in a smaller number of cases the disease was precociously malignant. The truth of this statement is borne out by the following table, which shows that the majority of cases were admitted in the second half of the first week. Only one of the fifty cases had received antitoxin before admission to hospital. This was a child who, after having had small doses of antitoxin at home on the fifth and sixth days of disease, was admitted to hospital on the seventh day and died on the eleventh day.

*Table V. Showing the Days of Disease on Admission to Hospital.*

1st day	.	.	0 cases
2nd "	.	.	7 "
3rd "	.	.	10 "
4th "	.	.	10 "
5th "	.	.	14 "
6th "	.	.	5 "
7th "	.	.	1 "
8th "	.	.	2 "
11th "	.	.	1 "

That the concomitant symptoms were severe is shown by the following facts. Fatal cardiac paralysis occurred in 19 cases, the signs of which first developed within a few days of the palatal affection. One case died of diaphragmatic paralysis on the fortieth day. Fourteen were hæmorrhagic cases, *i.e.* presented purpuric spots, with or without hæmorrhages from the mucosæ. Eleven of these were fatal. In all but one of the cases albuminuria was present. In 9 it persisted for three weeks or more. All but 6, or 80 per cent., of the survivors developed other paralyses, which are classified as follows: generalised paralysis, 8 cases; ocular paralysis, 14 cases; labial paralysis, 2 cases. The incidence of further paralysis among the non-precocious forms of palatal palsy was much less. Sixty-two cases (53·5 per cent.) occurred, only one of which, as already stated, was fatal.

Among the survivors, cardiac disturbance of some kind occurred in 14 cases. In 4 it was severe, and was associated with vomiting. Enlargement of the liver, a very grave sign, was present in 26 of the 50 cases. Seventeen of the 26 were fatal.

All the cases received antitoxin, but in spite of the massive doses which were injected the sequelæ were, as a rule, less marked than in milder cases in accordance with the law enunciated by the author that the frequency and intensity of serum phenomena are in direct relation to the size of the dose and in inverse relation to the character of the diphtherial attack. Babinski's sign, which Kiroff has recently noted in malignant diphtheria, was present in 5, or 51 per cent., out of 9 cases in which it was sought for. It is noteworthy that, as in Kiroff's cases, the extensor response co-existed with sluggish or absent knee-jerks. Among the thirty survivors, the duration of the paralysis considerably exceeded the average, being 43·3 days, as compared with 24·8 days, which was the average duration of the palsy in the 112 cases. In 3 cases the palatal palsy was short-lived, the duration in each case being six, seven, and eight days respectively. No other paralyses subsequently occurred in these cases.

In the majority of cases the paralysis, as is the rule in diphtheria, was incomplete, and was manifested only by a change in the voice. Regurgitation seldom occurred at a very early

stage, except in young children. Inspection of the fauces showed that the motility of the velum was only slightly impaired on phonation. In a few cases the palsy was unilateral, when it was subsequent to an angina that had been unilateral or predominant on that side.<sup>1</sup>

### *Diagnosis.*

During the first fortnight of the disease, especially during the second week, the sound of the voice should be tested daily in all severe cases. The preservation of a clear voice will be found to coincide with a normal heart, an absence of liver enlargement, and a good general condition; while a nasal twang shortly precedes or accompanies the signs of cardiac involvement and the apathy or restlessness usually associated with it. It is sometimes difficult to distinguish a nasal intonation from the thick character of the voice due to faucial cedema and abundant membrane. As a rule, however, a nasal voice does not develop till the cedema has subsided.

When regurgitation occurs early in diphtheria it must be distinguished from that due to mechanical obstruction produced by hyperæmia and faucial cedema, such as may occur in any form of sore throat. In diphtheria, though the membrane may still be present, the cedema has usually subsided by the time that regurgitation occurs.

### *Pathology.*

Maingault, the writer of the first monograph on diphtheritic paralysis, attributed the paralysis to a modification of nutrition of the palate under the influence of inflammation, and compared the paralysis of the palate to the similar phenomena that follow inflammation of the bladder and intestines. It was soon pointed out, however, that local inflammation did not account for cases of paralysis occurring where there had been no initial angina, nor for the paralysis attacking other parts than those which had been the site of membrane, *e.g.* the eyes. A purely local cause was therefore set aside in favour of systemic intoxication by Trousseau. The histological investigations of Charcot and Vulpian in 1862 in a fatal case of palatal paralysis showed

<sup>1</sup> Similar cases were reported by Gubler in 1861, by Gee in 1864, and more recently by Aubertin and Babonneix.

that the motor nerves alone were affected. Later observers, such as Oertel and Leyden, pointed out that other tissues besides the nerves were involved. Hochhaus, by his histological examination, proved that the morbid anatomy of paralysis of the palate was mainly an interstitial myositis. This view was adopted by Baginsky and Romberg, who distinguish early paralyses (Frühlähmungen) from post-diphtherial palsies. The former, according to them, arise from disease of the musculature, which in many ways is analogous to the change in the heart. Post-diphtheritic paralysis, on the other hand, depends on a degenerative change in the peripheral nerves, with occasional affection of the anterior cornual cells. The most elaborate histological researches yet published on the paralysis of the palate in diphtheria are those by Deguy, after researches carried on in Marfan's laboratory at the Hôpital des enfants malades.

Sections of the palate from cases that had died with early paralysis showed very marked inflammatory lesions. The presence of a large number of diplococci, both in the leucocytes and in the thrombosed capillaries of the part, made Deguy regard the condition as a diplococcæmia superadded to diphtheritic intoxication.

From this brief survey it will be seen that recent authorities agree with the early writers in regarding the affection of the palate as due to a local change. The extensive superficial necrosis of the fauces accounts for the unusually long duration of the paralysis, owing to the long time that elapsed before the tissues are completely regenerated. A further proof of the influence of the local inflammation in determining the palsy is furnished by the fact that ocular paralysis in diphtheria is never precocious. In the present series of cases it never started before the beginning of the fourth week, and sometimes was not noted till the sixth week, although the vision had been tested carefully at frequent intervals until then.

#### *Summary.*

1. Precocious palatal palsy in diphtheria is almost invariably associated with malignant forms, as is shown by the high mortality, the association of other grave symptoms during the acute stage, and subsequent more frequent development of paralysis in convalescence in the cases in which it occurs.

2. It resembles the ordinary forms of diphtheritic palsy in its tendency to be frequently incomplete, and by its higher incidence among young persons.

3. It is, as a rule, of much longer duration than the palatal affection which occurs at a later date.

## REFERENCES.

1. Aubertin. *Arch. gén. de méd.*, fév. 10, 1903.
2. Aubertin et Babonneix. *Gaz. des Hôp.*, 1902, p. 1285.
3. Babonneix. Nouvelles recherches sur les paralysies diphtériques," *Thèse de Paris*, 1904.
4. Baginsky. "Diphtherie und diphtheritischer Croup," 1898.
5. Berthelot. "De la gravité des paralysies diphtériques précoces," *Thèse de Paris*, 1904.
6. Deguy. *Rev. mens. des mal de l'enf.*, juin 1903.
7. Gee. *Med. Times and Gaz.*, 1864, p. 148.
8. Grancher, Bouloche, and Babonneix in Brouardel and Gilbert's "Traité de méd.", 2nd ed., 1905, art. "Diphtérie."
9. Gubler. *Gaz. méd. de Paris*, 1861, p. 704.
10. Henoch. "Lectures on Children's Diseases," *New Syd. Soc.*, 1889, vol. ii. p. 306.
11. Hochhaus. *Virchow's Archiv*, Bd. 124, S. 226, 1891.
12. Kiroff. *Rev. Neurol.*, Nov. 30, 1905 ; Abstract in *Review of Neurology*, Feb. 1906, p. 151.
13. Mackenzie. "Diphtheria," 1879.
14. Maingault. "De la paralysie du voile du palais à la suite d'angine," *Thèse de Paris*, 1854.
15. Marfan. *Bull. et Mem. de la Soc. méd. des Hôp. de Paris*, July 11, 1902 ; and "Leçons cliniques sur la diphtérie," 1905.
16. Moynier. "Compte rendu des faits de diphtérie dans le service de Trousseau," 1859.
17. Petit. *Rev. mens. des mal de l'enf.*, fév. 1897.
18. Rolleston, J. D. *Practitioner*, Nov. and Dec. 1904 ; *Ibid.*, May 1905 ; *M.A.B. Annual Reports*, 1904 ; *Review of Neurology*, Nov. 1905.
19. Romberg. "Lehrbuch der inneren Med.," 1905, art. "Diphtherie."
20. Ruault in "Traité de Méd.," par Charcot, Bouchard, et Brissaud, art. "Diphtérie."
21. Sanné. "Diphtérie," 1877.
22. Sevestre et Martin in Comby's "Mal de l'enf.," tom. 1, 1904, art. "Diphtérie."
23. Squire in Reynold's "System," vol. i., 1866, art. "Diphtheria."
24. Trousseau. *Union médicale*, 1851, p. 471 ; *Gaz. des Hôp.*, 1860, Nos. 1 and 5 ; *Clinique méd.*, 1st ed., 1861.
25. Variot. "La diphtérie et la sérumthérapie," 1898.



**THE PATHOLOGY OF GENERAL PARALYSIS.**

By DR HANS EVENSEN,

Medical Superintendent of Trondhjem Lunatic and Criminal Asylum

(Lecture delivered before the University of Christiania.)

**PART II.**

I HAVE dwelt so long upon the conditions of the vessels, especially of the cortical vessels, because they are conclusive with regard to the anatomical diagnosis of general paralysis, and upon those of the larger vessels, because they are very frequently somewhat neglected by writers on the subject. I now return to the lesions of the cerebral cortex.

*The neuroglia*

has increased much in general paralysis, and this increase will generally, though not necessarily, keep pace with the destruction of the nervous tissue. By neuroglia is here always meant all non-nervous tissue in the cortex outside the vessels. The biological limit between the tissue of mesoblastic and that of epiblastic origin is constituted by adventitia, according to Degenkolb, by the vascular neuroglial net according to Held, so that all tissue lying outside the adventitia is originated from the epiblast. According to the theory of Ford Robertson, a certain number of elements described by others as the "neuroglia" have developed from the mesoblast, and have their own special function ("mesoglia," as opposed to "neuroglia").

On the cortical surface will be seen a denser felting, mainly of coarse glial fibres, which may even penetrate into the pia mater. The filaments do not assume the direction of the original fibres in such a perfect manner as they do in typical cases of epilepsy (Alzheimer), and the breadth of the felting is more variable (Figs. 6-8). The glia fibres<sup>1</sup> may be traced downwards through the whole of the cortex. Especially along the vessels they may attain a considerable thickness. Even

<sup>1</sup> For daily work the methyl-violet method of Ford Robertson is very well adapted for staining glia in formalin-hardened preparations. Alcohol should not be used, as it is apt to destroy the finest fibres.

in the white matter, plexuses of densely interwoven fibres may be found.

Corresponding to this rich new formation of glial fibres, numerous retrogressively altered nuclei will be found in the outermost layer, recognisable by their shrunken form and deep staining in Nissl preparations; as a rule, much yellow pigment adheres to them. The most conspicuous objects in the outermost layer are the large *spider cells*, with their large pale nuclei, their distinctly stained and sometimes swollen cell body, and their numerous prolongations (Fig. 4). They may also be found in rows along the vessels, and it is now generally admitted that the protoplasmic processes are in part attached to the vessel wall by means of conical expansions. One side at least of the prolongation and the cone is often limited by a coarse glial fibre, which finally disappears in the glial network round the vessel, or ends in a fan-shaped termination. Through the whole cortex there will be seen many unusually large pale glial nuclei, with one or often several comparatively large nucleoli and a faintly perceptible protoplasm. Some of the glial cells are mere monsters, and the cell-body may exhibit the most curious forms, often resembling indistinctly limited areas, containing several nuclei (Nissl's "*Gliaraseen*"). Karyokinetic figures will be found comparatively rarely.

This proliferation of neuroglia seems to take place chiefly where the morbid processes are going on afresh, either in cases in which the disease is steadily progressive, or in those in which there are acute exacerbations during a chronic course; it will frequently be absent in slowly progressive cases. Spider cells have also been found, however, in paralytics who died during a remission.

The large pale (hyperplastic) nuclei will also be found in the white matter; besides these there are here many small dark nuclei resulting from the differentiation of the protoplasma into glial fibres.

According to the researches made by Ræcke, the proliferation of glia in the cerebellum occurs chiefly in the molecular layer—corresponding to the destruction of Purkinje's cells—also, but far less diffusely, in the granular layer, and least of all in the medullary layer.

In 1899 (see reference 51, p. 656), Nissl described as belong-

ing to the neuroglia some exceedingly long and narrow nuclei, occasionally slightly curved, which on a cursory view might be taken for proliferating endothelial nuclei, the more so as they generally lie near the vessels. Sometimes the pale nucleus, with a row of small nucleoli, will alone be seen ; but generally the cell-body may be perceived jutting out at the poles of the nucleus ; occasionally it will exhibit ramifications (Fig. 10). These "*staff cells*" (*Stäbchenzellen*), as Nissl now calls them, are considered of some importance in the diagnosis of general paralysis, as they will not be found so profusely in any other insanity. It is not improbable that these cells for the greater part are the same as the "staff-shaped" nuclei described by some earlier authors. In 1895, Harald Holm, a Norwegian, drew attention to the occurrence of these nuclei in masses in general paralysis (reference, p. 210). Whether they are really of neuroglial nature is perhaps doubtful. They will often be found close to the adventitia of the capillaries, and sometimes they may convey the impression of having something to do with the development of new capillaries.

The granulations of the *ependyma* were formerly considered to be due to a hyperplasia of the surface epithelium. Since a proper method of staining glia was introduced by Weigert, they have been found to be caused by proliferation of the subjacent glial tissue, accompanied by a differentiation of numerous fibres. The *ependyma* itself is not thickened, and in the sections it will often be lost on the tops of the prominences.

As a manifestation of the morbid process in general paralysis the hyperplasia of glia is generally admitted to be of a secondary nature. But still, everything is not said in the statement that owing to the degeneration of nervous tissue, nerve cells, and medullated fibres, there is nothing left to keep the glia in check. The fact is that this degeneration is not always followed by proliferation of glia.

#### *Nerve cells.*

With regard to the changes that affect the nerve cells in general paralysis, most authors, as if of one accord, do not deal with them minutely, pleading partly that the question is a very difficult one, partly that as yet no cell disease typical of general

paralysis has been found. It is no longer believed that the nerve cells react with a distinct anatomical change to each of the different kinds of noxious agents, which may disturb the equilibrium of its metabolism. There is no lack of variety in the reactions. Indeed, it would be a Sisyphean task to classify the changes into small groups (E. Meyer). Nissl has provisionally described eight different cell diseases, each of which may combine with one of the others; but even this grouping is not sufficient for all cases. To judge from the existing literature, however, the clue that leads to the recognition of these diseases still seems to remain in the Heidelberg laboratory. The ideas one could obtain from Nissl's description of them gave little more than the impression that the whole matter was exceedingly intricate. The common reluctance to grapple with the changes of the nerve cells still persisted. Since Nissl brought forward his equivalent theory, and since the artefacts produced by hardening as well as the post-mortem changes could be accurately recognised, it has no longer been possible to dismiss the question by saying that the image of the nerve cell in Nissl's preparations was an artificial one. It was then urged that the method was too subtle; it showed changes even where no insanity was present. Many pathologists, therefore, still did not make use of Nissl's staining, but kept to the old methods, which scarcely showed any result, and still called all the changes degenerations, which explained nothing. Others confined themselves to the examination of the large pyramidal cells, the conditions of which were least complicated, but which frequently, especially in general paralysis, were not altered at all. Among the changes here they dealt only with the chromatolysis, commencing at the centre, which scarcely made one any wiser. So it was a great relief when the neuro-fibrils were found, and attention was diverted from the stainable protoplasmic portion seen in the tedious Nissl preparation.

But still, the neuro-fibril methods are not very well adapted for use in anatomo-pathological examinations, and at present the question of changes in the nerve cells in the Nissl preparation cannot be evaded. Were it merely for facilitating mutual understanding it would be of great importance if all could agree with regard to some definite types of disease. In the following I refer to those types which Nissl has described.

In general paralysis the nerve cells are generally very much changed throughout the cortex, but not to the same extent in the different gyri, or in the different layers. Sometimes not a single cell will be found which has not been injured; in other cases the changes are slight. Most frequently the lamination of the cortex has been preserved; but the very fact that the morbid process has disturbed the arrangement of the cells suggests general paralysis (Nissl).

One of the most common cell diseases, and one that may most easily be recognised, is *chronic alteration*<sup>1</sup> (cell sclerosis). The cell shrinks and becomes darker, and at the same time the axis-cylinder process and the normally unstainable tracts take on the staining. The dendrites will be stained to a great extent; they become narrower and contorted. The contours of the cell have sharp edges. The *nucleus* becomes smaller, lengthened, and *angular*; the nuclear contents are so deeply stained that at last the nucleus cannot be distinguished from the rest of the cell.

Another alteration of the nerve cells which is easily recognised is the *severe form of cell disease*. Here the cell becomes faded, generally from the centre outwards. The cell-body becomes liquified. What is left of the stainable substance forms small circles, which are only slightly stained. *The nucleus becomes small and globular*; the nuclear contents are uniformly stained; the nucleolus becomes small and is removed towards the wall. The nuclear membrane and the axone show longest resistance.

*Acute cell disease* is not unfrequently met with. Its peculiar feature is that it attacks all the cells of the cortex simultaneously. The cell swells and becomes paler. The details are somewhat effaced, as the neuro-fibril tracts take on staining, and the chromatin bodies *moulder*. The prolongations of the protoplasm break down; the axone only shows strong staining. Later on the cell-body undergoes vacuolation. Even the *nucleus is swollen*; it moves closer to the margin of the cell, sometimes even beyond it; the nuclear contents do not take on staining, so that the nuclear network and the nucleolus stand out very clearly.

Of all cell diseases met with in general paralysis, these three

<sup>1</sup> There is no connection between acute and chronic cell disease and the acute or chronic course of general paralysis.

most frequently occur in a pure form as far as I have seen. The *granular decay* and the *cell shrinking* (*Zellenschwund*) seem to be less common. But there is scarcely one of Nissl's eight cell diseases which will not occasionally be found.

The different cell diseases will often be found simultaneously in the cortex, or even combined in the same cell. Thus an acute cell disease may occur in a chronically changed cell, so that the swollen nucleus does not appear round, and the diffusely-stained cell-body does not look quite pale, etc. Or a chronic and a severe cell disease may be combined. The combined forms are perhaps more frequent than the pure types, and they are often very difficult to distinguish. Further, the same cell disease may show a different appearance according to the stage of its development. Taking all this into consideration, one cannot but admit that the examination of nerve cells is a somewhat thankless task.

It is not easy to form any definite opinion as to which convolutions and which cell layers are most frequently attacked by these cell diseases. For that purpose a long series of carefully examined cases is required.

In cases of general paralysis which have lasted for several years, terminal stages of cell diseases and different forms of cell destruction will chiefly be found. The cells liquify, are demolished by vacuolation, or destroyed by glial cells, and finally disappear. Some remnants of the cells may remain, and here calcareous deposits will appear. These are not stained in the Nissl preparations. There may also be seen some peculiar incrustations, which stand out bluish-black in colour. Ultimately the cell remnants will break down into débris, or there will only be left a shadow of the cell, which retains its original shape (ghost cells).

In the cortex, areas may be seen in which scarcely any nerve-cells are left, and if the decay is irregular the mutual arrangement of the cells will be disturbed, as has already been mentioned. The spreading of the non-nervous tissue wherever the nervous tissue is destroyed will also help to disturb the cellular arrangement. The rows of cells will not only become more open, but they will be interrupted. In other insanities where a considerable decay of nerve cells occurs, the normal order of the cells, with their tips towards the surface of the convolutions, has been

preserved, even if the rows have become less crowded. Something like this may be seen only in serious forms of arteriosclerosis, as far as I know. Weber, however, relates that he has seen the same condition in epileptic insanity.

In some places it is chiefly the intercellular fibrillar substance, the *grey reticulum* ("the nervous grey," Nissl) which has been destroyed, so that the cells stand close up to each other. At first sight it therefore looks as if the cells in the cortex had increased in number.

The same changes of nerve cells as in the cortex will be found in the nuclei of the medulla oblongata.

Since Exner's (1881), and especially since Tuczek's (1884) researches, we have known that large numbers of

#### *Medullated nerve fibres*

are destroyed in general paralysis. In consequence of the total decay of nervous tissue in slowly progressing cases the cortex has, in general, shrunk and become narrower.

The disintegration of medullated fibres begins with the tangential fibres of the surface, which may, however, in some cases be preserved. It will decrease gradually from the surface of the cortex towards the white matter. The fibres will be destroyed in greatest number in the pyramidal cell layer, then in the layer of the granules (Baillarger's bundle); but the interradianal network also will become more sparse (Kaes, Mott). Frequently the myeline sheath only has disappeared, so that the axis-cylinder is almost bare (Kaes). The waste of fibres is most extensive in those gyri, which, on the whole, are chiefly attacked; it is almost always considerable, even in the islands of Reil, and may be traced in all parts of the brain. The decay will very seldom be confined to small spots (Siemerling, in the work of Cramer, Binswanger). While all the destruction of medullated sheaths which is in full activity may be recognised with some accuracy by Marchi's method,<sup>1</sup> it is rather difficult to decide in a completed process whether, if there is no considerable loss, any fibres have been lost or not. On the one hand the distribution of tangential fibres is unequal, and their quantity varies with the individual;

<sup>1</sup> With regard to the fallaciousness of this method see Nissl (in "Encyklopädie der mikroskopischen Technik," p. 962) and Spielmeyer ("Die Fehlerquellen der Marchi'schen Methode," *Centralblatt. für Nerv. u. Psych.*, 1904, p. 757).

on the other, the method (Weigert's, but still more Pal's modifications of the same) is not at all mathematically exact.<sup>1</sup> Exner's method is, no doubt, more reliable, but it is "not suitable for general use" (Weigert).

In certain conducting tracts, even in their course into the spinal cord, secondary destruction of fibres will occur as a consequence of the focal lesions. As a rule these focal lesions are not very well marked in general paralysis, as they are due to a more delicate lesion of the cortex, and they will soon disappear, unless that part of the cortex is completely destroyed (Lissauer). It is a well-known fact that the focal lesion, indicated by the clinical symptoms, may be looked for in vain in the post-mortem examination. The gross circumscribed lesions, such as hæmorrhages and softenings, exhibit nothing characteristic of general paralysis.

### PART III.

#### *Anatomical differential diagnosis.*

The tissue-changes of the cerebral cortex, which have now been described, have all one feature in common: they occur diffusely, although they may attack certain localities in particular. The hyperplasia of neuroglia, and for the greater part the destruction of medullated fibres, are the consequences of the changes of nerve cells; these have most likely been directly injured by the pathogenic agent, but the changes in themselves are not characteristic of general paralysis. The only diffuse anatomical sign which to a certain degree in itself characterises the process as due to general paralysis, is the infiltration by plasma cells of the adventitial lymph spaces. It does not seem very probable, however, that the vascular change is the primary lesion in the morbid process, as many authors have maintained (Magnan, L. W. Weber, Köppen, Tschisch, Nageotte, Angiolella, and others), nor that this change is the source of the alterations in the nervous tissue. The vessels as well as the nerve cells, perhaps to some extent even the medullated fibres, may probably all be directly affected by the toxic agent.

As far as we know at present, the dense aggregation of

<sup>1</sup> The preparation is not always evenly penetrated by *chrome*, or it will be too far differentiated; in formalin-hardened preparations the after-hardening in alcohol has a deleterious effect upon many fibres.



plasma cells in the lymph space must be considered as a peculiar *inflammatory condition*, and in general paralysis this, I repeat, is diffuse. This is very important with regard to the anatomical differential diagnosis of general paralysis. The consequence of this is simply that Klippel's merely "degenerative paralysis" (which partly is identical with what others call pseudo-paralysis), and Binswanger's transitional forms without inflammations, do not belong to general paralysis. It is this sign also which makes it possible microscopically to distinguish general paralysis from similar changes in arterio-sclerotic insanity, chronic alcoholism and epilepsy, and from non-inflammatory forms of cerebral syphilis, where there are widespread changes without adventitial infiltrations (Alzheimer, Nissl).

Consequently the opinion can no longer be upheld that the capillary lesions are alike in both general paralysis and in *senile insanities*, and that there are forms of senile dementia which cannot be anatomically distinguished from general paralysis. If there is a widespread infiltration by plasma cells, the disease is a senile paralysis. It is unnecessary here to point out other distinctive features. According to Alzheimer, senile paralysis holds a special position, as the inconsiderable proliferation of neuroglia in this disease is not in proportion to the extensive decay of nervous tissue. Clinically also there is some difference from the ordinary general paralysis, as dementia—not paralytic symptoms—is the most prominent feature of senile general paralysis.

The insanities in drunkards are often simply called *alcoholic*. This is so even in cases in which the clinical symptoms afford evidence of general paralysis, but in which the usual gross meningeal changes have not been revealed by autopsy. Several cases which came under my observation labelled chronic alcoholism turned out, under microscopical examination, to be general paralysis. The diagnostic difficulties have induced several authors to establish an alcoholic paralysis as an intermediate form. In a previous paper (p. 61) I stated that after some time the disease will manifest itself either as general paralysis or as an alcoholic insanity (pseudo-paralysis). I added, however, that if death occurred before the diagnosis could be clinically determined, even autopsy could not always settle the question. This problem, which pathological anatomy

could not solve at that time (1899), it has now been able to work out. In alcoholic insanities no inflammatory infiltration will be found.

Even epileptic fits occurring in general paralysis, especially in status epilepticus and confusional states, leading to death within a short time, will sometimes be clinically taken for *epilepsy*, while the microscopical examination will prove it to be general paralysis.

There are, however, some acute and subacute forms of non-purulent *encephalitis*, in which the cell infiltration is no longer the criterion, and then the question must be settled by the process as a whole, *i.e.* affected nerve cells, proliferation of neuroglia, abundance of staff cells, loss of medullated fibres, and at the same time infiltration of the vessel sheaths and development of new vessels as described above. Special importance must be attached to the extent of the process. As far as is known at present the *encephalitis* is always a circumscribed lesion. The same distinction will apply to cases of epilepsy and idiocy caused by *encephalitis* (Degenkolb, Nissl). From a clinical point of view these cases will hardly give rise to confusion. For the same reason some other conditions, in which plasma cells occur, need not here be taken into consideration.

It is far more difficult, though not impossible, to distinguish general paralysis from inflammatory forms of *cerebral syphilis* (gummatous meningo-*encephalitis*). Here the same cell forms will be found in the infiltration, but the process does not seem to spread over the whole cortex as in general paralysis; it is most intense in the immediate surroundings of the specific new growths or surface inflammations. Some convolutions may be left quite uninjured. If gummata are found it will perhaps be most correct to call the disease cerebral syphilis, even if it differs in no other respect from what is commonly seen in an ordinary general paralysis. It may be, however, that such a case, which will seldom be met with, represents a combination of both diseases. According to my opinion, Heubner's endarteritis belongs to the antecedent syphilis, and does not prove that the disease in question is cerebral syphilis and not general paralysis. Along with the syphilitic meningo-*encephalitis* the so-called gummatous surface inflammation will always be found in the meninges of the spinal cord (Nissl); but it is not certain

that this inflammation may not also be found in general paralysis.

Our next task will be to discuss the

### *Etiology*

of the inflammatory condition. I shall here confine myself to mentioning the contributions which up to this time pathologists have made towards the solution of this problem.

It might seem to be an obvious conclusion that the paralytic changes were immediate consequences of syphilis. Previous syphilitic infection will be found as often amongst the antecedents of general paralysis as of extra-cerebral diseases of undoubted syphilitic origin. It will almost always be found in juvenile general paralysis, and the possibility of its existence cannot be left out of account even in cases in which the history does not affirm it. That paralytics may as a rare exception acquire recent syphilis does not disprove a previous infection. At present such a case will not even be accepted as general paralysis, unless the diagnosis is verified by microscopical examination. Further, general paralysis may be combined with lesions which anatomically present themselves as being of specific syphilitic origin, and on the other hand syphilis may bring about changes, the anatomical features of which do not reveal any specific nature.

Against the theory (maintained by Strumpell, Möbius, Hirschl, and several others) that general paralysis is a simple after-effect of syphilis, many arguments have been brought forward: the differences, clinical as well as anatomical, between general paralysis and ordinary cerebral syphilis, the negative results of anti-syphilitic treatment, the steadily progressive malignant course of the disease, and finally the absence of general paralysis in several localities in which syphilis is a common occurrence. At all events the said theory would have to be altered in so far as it maintains that general paralysis is quite a special form of syphilis.

It must at least be regarded as highly probable that syphilis is a *necessary* condition for the onset of general paralysis, just as poisoning by alcohol is necessary for the appearance of delirium tremens. Drinking and head injuries which often supervene cannot be supposed to have any other effect than to provoke

or accelerate the outbreak; nor can they—any more than exhaustion (Binswanger)—account for the anatomical picture of the disease. The changes in the cerebral cortex indicate that general paralysis is the outcome of intoxication. But if we desire to discover what then supervenes on the syphilis, and so gives rise to general paralysis, we must not confine ourselves to the examination of the brain and the spinal cord. For general paralysis is not a disease of the nervous system only, although here the changes are most obvious. The lesions of the other organs are no mere consequences of the alterations of the brain and of the spinal cord. The whole vascular system is affected, the bactericidal properties of the blood have been lost or reduced (Idelsohn); its auto-virulence has increased (D'Abundo). The kidneys are not sound (Angiolella); there will be found abdominal and intestinal catarrh, and very frequently also fatty liver. Diminution of bone marrow, fragility of bones, degenerative changes of the ear cartilage, liability to bed-sores, are all common symptoms. There may be observed fluctuations in the state of nutrition and temperature, which cannot be accounted for by the changes in the casual condition of the patient. There can, therefore, be no doubt whatever that general paralysis is a general disease. Kraepelin regards it as a *general disease of metabolism* akin to myxœdema, diabetes, dementia præcox, and other diseases. According to this author, it may have various sources, but it develops chiefly on the basis of syphilis, and will in its turn cause the special paralytic changes of the nervous system.

On account of the pre-supposition that intoxications are the common basis of insanities, the Italian as well as the younger Scotch alienists have instituted *bacteriological* examinations in paralysis. Of course no weight can be laid upon occasional infections in the terminal stage, caused by bed-sores, by cystitis (D'Abundo), or by pneumonia; nor can any importance be attached to certain post-mortem discoveries of bacteria (*e.g.* the coli bacillus). Cultures have been taken from the urine (coli bacilli, Grimaldi), from the blood (streptococci, *ibid.*), and from the cerebro-spinal fluid ("bacillus viscosus," Montesano and Montesori). The streptococci were not really taken into consideration as a cause of the disease; the coli bacillus was considered somewhat more important. Lewis Bruce observed

that the blood serum of paralytics manifested an agglutinative power on the coli bacillus during remissions of the disease, but not while the paralysis was active, and from this fact he derived the conclusion that there might exist a causal connection between this bacillus and general paralysis. E. Raimann, however, stated that the reaction of the agglutination was too irregular to permit of any conclusion being drawn from it; according to his experiments, normal blood serum has an even greater agglutinative action on the coli bacillus than that of paralytics.

Lately attention has been drawn to the diphtheria bacillus by the systematic experiments made by Ford Robertson, assisted by M'Rae, Jeffrey, and Shennan. They demonstrate that an invasion of micro-organisms takes place in the mucous membrane of the respiratory tracts and the alimentary canal, probably because there is a general as well as a local weakening of the normal defences. As the resistive powers are weakened, the parasites will develop a pathogenic action. Coli bacilli as well as streptococci may be found; but the micro-organism to which the principal importance is ascribed is a bacillus which resembles the Klebs-Löffler diphtheria bacillus in all respects except in its virulence. It can be cultivated from the respiratory tracts in living paralytics, but not from the blood; after death, pure cultures can be obtained from the surface as well as from the muscular coat of the stomach and of the small intestines, from mouth, bronchi, and lung tissue, sometimes also from the brain, but probably only when a general blood infection has taken place. Chiefly by using a modification of Neisser's method, the bacillus may be identified in sections of the catarrhal exudation from the organs mentioned above, except from the brain. In the lymphatics of the walls of the same tracts filamentous organisms have been found, which probably are the bacilli in some form of involution, perhaps occasioned by the presence of anti-bodies in the serum. Experiments with anti-diphtheria serum have had no effect. Guinea-pigs have proved insusceptible to the bacillus. In rats fed on bouillon cultures the organism produces a fatal disease with symptoms from the nervous system; and the anatomical changes of vessels, meninges of the brain, and neuroglia, as well as those in the respiratory and alimentary tracts, "have a distinct resemblance to those that occur in general paralysis."

Ford Robertson, therefore, goes further than to establish the regular occurrence of a diphtheroid organism in the respiratory and alimentary tracts in cases of general paralysis in asylums. Although a parasitic infection might be very dangerous to a paralytic individual, this bacillus is not merely a parasite. According to Robertson, general paralysis is the result of a chronic toxic infection brought on by the invasion of those tracts by *various* bacterial forms ; but it is a Klebs-Löffler bacillus of modified virulence which mainly gives the disease its special paralytic character.

While I was working in the laboratory of the Scottish Asylums for some months of the winter of 1903, I had an opportunity of observing the pure cultivation of this bacillus. It must be admitted that the bacillus, stained by Neisser's method, showed the typical appearance of the Klebs-Löffler bacillus. Still I cannot help thinking that for the present it is perhaps advisable not to insist upon the identity of this diphtheroid bacillus with the true diphtheria bacillus. There are, indeed, essential features of the life of the diphtheria bacillus with which we are not familiar ; and as long as experiments on animals have not demonstrated that the poisonous effect of the diphtheroid bacillus can at all be neutralised by anti-diphtheritic serum, we are not warranted in identifying the two bacilli. In my opinion the supposition that the diphtheroid bacillus shares the responsibility of causing insanity with other bacteria, the importance of which would thus be quite indefinite, merely complicates the hypothesis. The only advantage that, perhaps, may be derived from this supposition, is that the inefficacy of the anti-diphtheritic serum might be imputed to the mixed infection. But we cannot very well imagine more than *one* active agent in an anatomical process which is so constant in its occurrence, and which, in spite of its multiformity, has such a peculiar character as general paralysis. Finally, this hypothesis pre-supposes a still unknown factor, which weakens the natural power of resistance, not only against the attacks of micro-organisms in general—as by such generalities nothing definite would be explained—but against this diphtheroid bacillus in particular.

Now there is nothing to prevent us assuming that this unknown factor may be syphilis, and Robertson's hypothesis

would then more easily find favour with those who believe that the way to general paralysis lies through syphilis. According to Klippel, the "inflammatory forms" described by this author are due to a "trivial" infection, the occurrence of which is also facilitated by a previous syphilis. But then, again, if a previous syphilitic infection is necessary, it would be difficult to explain the fact that in animals the diphtheroid bacillus can by itself give rise to anatomical changes which resemble those in general paralysis, all the more so if the bacillus should prove to be identical with the ordinary diphtheria bacillus, which up till now has proved innocuous to rats. The chief interest will be concentrated in these experimental changes, of which only preliminary communications have as yet been made.

It will be a point of importance to ascertain whether plasma cells do or do not occur, even if the conditions for their occurrence might be different in the experimental animals from what they are in human beings. But even when the results of experiments in animals harmonise, to a great extent, with the anatomical picture of general paralysis, it would be premature to conclude that the processes are identical. In an insane dog, Nissl once found changes which could not be distinguished from those which are characteristic of general paralysis.

It is to be hoped that these investigations will be carried on, and that other pathologists will take them up. Theoretical criticism is of no value, and the examination of the cerebral cortex alone is not likely to solve the problem. One thing ought to be considered an established fact: that the pathology of general paralysis can only be established by investigations which are founded on the doctrine that this insanity is a general disease. Whether one had better search for a micro-organism or lay stress upon inquiries into the metabolism is a secondary question. All who trust that one day or other we shall succeed in bringing psychiatry into line with general medicine have in general paralysis a field for research in which they may expect to reap fruit of their labour.

---

Gentlemen, since general paralysis was established as an insanity proper, eighty years passed before we learned to make the diagnosis from the microscopical picture. That this insanity

has been the first of which we have a pathological anatomy is due to its frequency, to the fact that its clinical picture is easily recognised, and to the gross anatomical changes which invite minute examinations. That this result has been obtained must, however, be ascribed chiefly to the fact that so many workers have taken part in the investigation. It has certainly been of importance also that contributions to the anatomy of the disease have come from various sources. Most of the researches have, of course, been made by alienists, as they almost alone have the opportunity of studying insanities. The development of psychiatry lies in their hands, theirs is the duty therefore of working scientifically for the profession. An acknowledgment of this duty is represented by the founding of laboratories for each asylum, or for several asylums in common. Here the alienist who takes a particular interest in pathological anatomy may concentrate his energy on this matter; here the novice may be instructed. That these laboratories abroad give admission to foreigners is a great benefit to those who live far from the centres of culture and science. I for one have benefited greatly thereby, and I should not like to conclude this paper without having expressed my sincere gratitude for the cordiality with which I was met during my studies at the laboratory of the Psychiatric Clinique in Heidelberg, as well as at the laboratory of the Scottish Asylums in Edinburgh. I wish also to emphasise the fact that these laboratories do not render it unnecessary for the assistant physicians of the asylums to take part in the anatomical investigations. This is, in fact, the only way in which it will be possible for them to keep up their knowledge of the literature bearing upon this subject. Nobody expects that great discoveries will be made in this way. It is, however, of great importance as regards the steady development of this science that the daily minor work in the laboratory should be carried out by a sufficient number of investigators. Alienists in general ought not to consider anatomy as outside their psychiatric training, as they have done up till now in this country as well as abroad. In countries where there are no institutions for the teaching of beginners in this subject, it is of still more importance that those who occupy superior positions in asylums should be able to instruct their assistants in the principles of the subject, and thus to lay the foundation of their further training. The



right distribution of the work need not therefore be affected. What is not actually necessary "for household use" may be left to those who take special interest in pathological anatomy. But in every asylum it ought to be expected of the assistant physicians that they should at least be able to make that test of general paralysis which consists in the demonstration of plasma cells.

## REFERENCES.

1. D'Abundo. *La Psichiatria*, 1889. Cited by No. 63.
2. Almkvist, Johan. "Beiträge zur Kenntniss der Plasmazellen, insbesondere beim Lupus," *Arch. f. Dermatologie u. Syphilis*, Bd. 58, 1901, S. 105, 110.
3. Alzheimer. "Ueber die anatomische Ausbreitung d. paralytischen Degenerationsprozesses," *Neurol. Centralblatt*, 1896, Oktober, S. 1007.
4. ——— "Beitrag z. pathol. Anatomie der Hirnrinde u. zur anat. Grundlage einiger Psychosen," *Monatsschr. f. Neurologie u. Psychiatrie*, ii. S. 82.
5. ——— "Ueber atypische Paralyse," *Monatsschr. f. Psych. u. Neurol.*, xi. S. 73.
6. Angiolella. "Di alcuni problemi sulla paralisi progressiva e dei piu recenti lavori su di essa," *Il manicomio moderno*, vol. 10, 1894, No. 3.
7. Anglade. See the discussion on Paralysis at the Brussels Congress, 1903, *Revue neurol.*, 1903.
8. Bayle. "Traité des maladies du cerveau et de ses membranes," 1826.
9. Binswanger. "Die Abgrenzung d. allg. progr. Paralyse," *Berlin. klin. Wochenschr.*, 1894, S. 1103; *Allg. Zeitschr. f. Psych.*, Bd. 52, S. 488.
10. ——— "Die patholog. Histologie d. Grosshirnrindenerkrankung bei der allg. progr. Paralyse." Jena, 1903.
11. ——— "Beiträge zur Pathogenese u. differentiellen Diagnose der progr. Paralyse," *Virchows Archiv*, Bd. 154, 1898, S. 389.
12. Boedecker u. Juliusburger. "Anatomische Befunde bei Dem. paral.," *Neurol. Centralbl.*, 1897, S. 774.
13. Bruce, Lewis. "Clinical and Experimental Observations upon General Paralysis," *Brit. Med. Journ.*, 1901, p. 1600.
14. Calmeil. "De la paralysie considérée chez les aliénés." Paris, 1826.
15. Cramer. "Pathologische Anatomie der Psychosen," "Handbuch d. pathol. Anat. des Nervensystems," S. 1486, note.
16. Degenkolb. "Beiträge z. Pathologie der kleinen Hirngefässe. Ueber Vorkommen intraadventitiellen Infiltrate der Rindengefässe bei diffusen Rindenkrankheiten," *Allg. Zeitschr. f. Psych.*, 59, 1902, p. 714.
17. Donath. "Das Vorkommen u. die Bedeutung des Cholins, etc.," *Zeitschr. f. physiol. Chemie*, 1903, Bd. 39, p. 526.
18. Dupré. "Psychopathies organiques," "Traité de pathol. mentale" (Ballet). Paris, 1903, p. 115.
19. Evensen. "Den kron. alkoholismes kliniske former," *Norsk mag. f. lægev.*, 1899, No. 2.
20. ——— "Vascular Lesions in Mental Diseases," *Transactions of the Medical Society in Kristiania*, 1901, p. 186.

21. Friedmann. "Zur Lehre, insb. z. pathologischen Anatomie der nicht eitrigen Encephalitis, *Deutsche Zeitschr. f. Nervenheilk.*, Bd. 14.
22. Gaupp. "Neuere Arbeiten über die progressive Paralyse d. Irren," *Monatsschr. f. Psych. u. Neurol.*, 1897, Bd. i. p. 255.
23. Del Greco. "Sulle alterazioni delle pie meningi cerebrali negli alienati," *Rivista speriment.*, vol. xvii., 1891.
24. Grimaldi. *Il manicomio*, 1896, 2-3. Quoted by No. 60.
25. Havet. "Des lésions vasculaires du cerveau dans la paralysie générale," *Bull. de l'acad. de méd. de Belgique*, 1902.
26. Held. "Ueber den Bau der Neuroglia und über die Wand der Lymphgefäße in Haut und Schleimhaut." Leipzig, 1903.
27. Heubner. "Die luetischen Erkrankungen der Hirnarterien." Leipzig, 1874.
28. Hewitt, Prescott. *London Med.-Chir. Transactions*, vol. xxviii., 1845. Quoted by No. 26.
29. Huguenin. "Pachymeningitis int. hæmorrh." In Ziemssen's "Handbuch," 1878, xi. 1, p. 384.
30. Idelsohn. "Ueber das Blut u. dessen Bactericides Verhalten bei prog. Paralyse," *Arch. f. Psych.*, Bd. 31, p. 640.
31. Joffroy and Mercier. "De l'utilité de la ponction lombaire pour le diagnostic de la paralysie générale," *Revue de neurol.*, 1902, p. 825.
32. Jores. "Wesen u. Entwicklung der Arteriosklerose." Wiesbaden, 1903.
33. Kaes. "Rindenbreite u. Markfaserchwund bei allg. Paralyse," *Wiener medicin. Wochenschr.*, 1900.
34. ——— "Zur patholog. Anatomie d. Dementia paralytica," *Monatsschr. f. Psych. u. Neur.*, 1902.
35. Klippel. "Les paralysies générales progressives." Paris, 1898.
36. ——— "Histologie de la paralysie générale." Congrès de Bruxelles, 1903, *Revue neur.*, 1903, p. 814.
37. Lissauer. "Sehhügelveränderungen bei prog. Paralyse," *Deutsche med. Wochenschr.*, 1890, No. 26.
38. ——— bei Storch. "Ueber einige Fälle atypischer progressiver Paralyse," *Monatsschr. f. Psych. u. Neurol.*, Bd. 9, 1901, p. 401.
39. Mahaim. "De l'importance des lésions vasculaires dans l'anatomie pathologique de la paralysie générale et d'autres psychoses," *Bullet. de l'académ. de méd. de Belgique*, 1901, juli, p. 600.
40. ——— "L'importance diagnostique des lésions vasculaires dans la paralysie générale," *Ibid.*, 1902.
41. v. Marschalkó. "Ueber die sogenannten Plasmazellen, etc.," *Arch. f. Dermatologie u. Syph.*, Bd. 30, 1895, pp. 3, 241.
42. "Die Plasmazellen in Rhinoskleromgewebe, etc.," *Ibid.*, Bd. 54, 1900, p. 235.
43. ——— "Zur Plasmazellenfrage," *Zentralbl. f. allg. Path. u. path. Anatomie*, Bd. 10, 1899.
44. Mendel. "Die progressive Paralyse d. Irren." Berlin, 1880.
45. Meyer, E. "Die pathol. Anatomie der Psychosen." Orth—Festschrift.
46. Meyer, L. "Die allg. progr. Gehirnlahmung eine chron. Meningitis," *Charité-Annalen*, 1858.

47. Meyer, L. "Die pathol. Anatomie der Dementia paralytica," *Virchow Archiv*, 58, 1873, p. 292.
48. Montesano e Montessori. "Ricerche batteriologiche, etc.," *Riv. di Psicol., Psichiat., Neuropatologia, etc.*, 1897, 1, 15. Ref. *Neurol. Centralbl.*, 1898, p. 549.
49. Mott. "Observations upon the Etiology and Pathology of General Paralysis," *Arch. of Neurology*, 1889, i.
50. Nissl. "Sind wir imstande, aus dem pathol.-anat. Befunde die Diagnose der progr. Paralyse zu stellen?" *Monatsschr. f. Psych. u. Neurol.*, iv.
51. ——— "Ueber einige Beziehungen zwischen Nervenzellenerkrankung u. glösen Erscheinungen bei verschiedenen Psychoosen," *Arch. f. Psych.*, Bd. 32, H. 2.
52. "Die Diagnose d. progr. Paralyse," *Neurol. Centralbl.*, Bd. 21.
53. ——— "Ueber einen Fall von Geistesstörung bei einem Hund," *Centralbl. f. Nervenheilk.*, 1890, Cfr. *Arch. f. Psych.*, Bd. 33, H. 2.
54. "Kritische Bemerkungen zu Schmaus: Vorlesungen über die pathol. Anatomie des Rückenmarks," *Centralbl. f. Nerv. u. Psych.*, 1903, p. 88.
55. ——— "Die Bedeutung der Lumbalpunktion," *Centralbl. f. Nerv. u. Psych.*, 1903, p. 225.
56. Oppenheim. "Die syphilitischen Erkrankungen des Gehirns," 1903, p. 113.
57. Orr and Cowan. "A Contribution to the Morbid Anatomy and the Pathology of General Paralysis of the Insane," *Journ. of Ment. Sci.*, 1900, p. 688.
58. Pappenheim. "Wie verhalten sich die Unna'schen Plasmazellen zu Lymphozyten?" *Virch. Arch.*, Bd. 165 (1900, p. 365), Bd. 166 (1901, p. 424).
59. Raacke. "Ueber Gliaveränderungen im Kleinhirn bei progr. Paralyse," *Arch. f. Psych.*, Bd. 34.
60. Raimann. "Zur Aetiologie der progr. Paralyse," *Wiener klin. Wochenschr.*, 1903, No. 13.
61. Rehm. "Einige neue Färbungsmethoden zur Untersuchung d. central. Nervensystems," *Münchener med. Wochenschrift*, 1892, p. 217.
62. Robertson, Ford. "A Text-book of Pathology in relation to Mental Diseases." Edinburgh, 1900.
63. Robertson, M'Rae, and Jeffrey. "Bacteriological Investigations into the Pathology of General Paralysis of the Insane," *Rev. of Neurol. and Psych.*, 1903, No. 5.
64. Robertson, Ford. "Histological Evidence of the Presence of an Organism resembling the Klebs-Löffler Bacillus in Cases of General Paralysis of the Insane," *Rev. of Neurol. and Psych.*, July 1903.
65. ——— "The Pathology of General Paralysis of the Insane," *Brit. Med. Journ.*, Oct. 24, 1903.
66. Straub. "Gefäßveränderung bei allg. Paralyse." *Verh. d. Gesellsch. deutscher Naturforscher u. Aerzte. München*, 1899. Ref. *Neurol. Centralbl.*, Bd. 20, p. 957.
67. Tuczek. "Beiträge zur Anatomie u. Pathologie d. Dementia paralytica." Berlin, 1884.
68. Unna. "Ueber Plasmazellen, insbesondere bei Lupus," *Monatsschr. f. prakt. Dermatologie*, Bd. 12, 1891.

69. Unna. "Ueber die Bedeutung der Plasmazellen für die Genese der Geschwülste der Haut," etc., *Berl. klin. Wochenschr.*, 1892, No. 49.
70. ——— "Plasmazellen," "Encyklopädie der mikrosk. Technik," 1903, ii. p. 1116.
71. Vogt, Ragnar. "Das Vorkommen von Plasmazellen in der menschlichen Hirnrinde, etc.," *Monatsschr. f. Neurol. u. Psych.*, 1901, p. 211.
72. Waldeyer. "Ueber Bindegewebszellen," *Archiv f. mikroskop. Anat.*, Bd. 11, 1875.
73. Weber, L. W. "Ueber die sogen. gallopiierende Paralyse nebst einigen Bemerkungen über Symptomatologie u. pathologische Anatomie," *Monatsschr. f. Psych. u. Neur.*, Bd. 14, 1903, pp. 374, 450.
74. Weigert. "Beiträge zur Kenntnis d. normalen menschlichen Neuroglia." Frankfurt, 1895.

## NOTE TO REFERENCES (IN FINE).

After this paper had been written (September 1904) the first volume appeared of "Histologische u. histopathologische Arbeiten, etc.," herausgegeben von *Franz Nissl*, Jena, 1904, containing: "Histologische Studien zur Differentialdiagnose der progr. Paralyse" by *Alzheimer*, and "Zur Histopathologie der paralytischen Rindenerkrankung" by *Nissl*. As the lecture above rendered was delivered immediately after the paper was written, there was no time to take advantage of these important treatises by *Alzheimer* and *Nissl*.

---

## Abstracts

### PHYSIOLOGY.

**THE MOTOR AREAS IN THE CEREBRAL CORTEX OF (342) DASYURUS VIVERRINUS.** J. F. FLASHMAN, *Reports Path. Lab. of Lunacy Depart., N.S.W.*, Vol. i., Part 2]

THE motor centres in the cerebral cortex of this marsupial, the "native cat" of New South Wales, were investigated by electrical stimulation in several specimens. The convex surface of the hemisphere is marked by only one fissure, the sulcus orbitalis, which is generally simple, but there is occasionally an apparent bifurcation of its upper end. The posterior branch is, however, not a true sulcus, but only a groove in which an artery lies.

Movements were obtained only by stimulation of the posterior lip of this sulcus. With a minimal current the movement was crossed, but with a slight increase of the current the limbs of the same side were also affected. The areas for the various groups of muscles overlap one another to a very large

extent, and there are in addition well-marked centres for very definite associated movements.

The arrangement of the motor centres from above downwards is—leg, arm, the movement of seizing with the mouth and claws an object in front and to the opposite side, snarling, and, at the inferior end of the fissure, the movements of the tongue and jaw.

GORDON HOLMES.

### **PATHOLOGY.**

**DENDRITES AND DISEASES.** Sir WILLIAM GOWERS, *Lancet*, (343) July 14, 1906, p. 67.

In a lecture delivered at Queen's Square, Sir William Gowers deals with some of the remote implications of the neurone doctrine. He first describes the facts, as given in the current text-books, on which the neurone theory rests. These are mainly findings in histology. He regards the continuity of the ultimate fibrillar network of the dendrites as improbable, and holds that Cajal's teaching of "contiguity not continuity" is borne out by observation. The significance of the fact that neurofibrils never end in nerve cells has been too much overlooked, yet it disposes of the old view that the nerve cell is the source of nerve impulse. In speaking of the contractility of the dendritic terminations, he suggests the analogy between the hyaloplasm of the fibrils, and that of muscular fibres. The observations that have been made on dendritic retraction during hibernation are then referred to, with their bearing on the theory of sleep. The author claims that the isolation thus brought about of the higher structures from the lower structures that continually excite them allows of rest of the former, so that nutritional renewal may take place, and that this view is a more satisfactory explanation of the phenomena of sleep than any other physiological one.

Turning to disease, one may look upon chorea as a nutritional disturbance of the cortical dendrites (see *Review of Neurology*, March 1906, p. 219). Paralysis agitans may be looked upon as a senile dendritic disease. This would explain why researches made upon the motor cells have been made in vain, and why no degeneration occurs in the pyramidal fibres. An attack of epilepsy may be regarded as the passage of a special form of excitation through the dendritic fibrillæ of the cortex with an extension almost explosive in its rapidity. Again, hysterical hemianæsthesia may be due to a mechanism similar to that occurring in sleep, so that the impulses of pain cannot reach those neurones that subserve consciousness.

ERNEST JONES.

**THE NON-OCCURRENCE OF AUTOGENOUS REGENERATION  
(344) OF NERVE FIBRES.** (Ancora un' esperienza contro l'auto-  
rigenerazione delle fibre nervose.) E. LUGARO, *Riv. di Patol.*  
*nerv. e ment.*, 1906, p. 273.

LUGARO records the results of experimental observations on three puppies in which he extirpated the lumbo-sacral portion of the spinal cord, together with the corresponding spinal ganglia. The animals were kept alive for periods varying from two and a half to three months. They were then killed and the nerves of the lower limbs examined, not only with the osmic acid method but also by Cajal's new axis-cylinder process with reduced silver.

He found no evidence of regeneration in any of the nerves, with the exception of one case, in which five or six myelinated fibres were present and appeared to be traceable to a spinal ganglion which happened to have been incompletely removed.

If the proximal part of the nerves be not extirpated, non-myelinated fibres may appear in the peripheral stump, these apparently being derived from the sympathetic nervous system. All the new fibres which arise from the sympathetic ganglia and grow into the peripheral nerves are non-myelinated. Lugaro also states that regeneration of striated muscle may occur autonomically, even when all nervous influence, cerebro-spinal or sympathetic, is withdrawn.

PURVES STEWART.

**ON THE DEGENERATION OF NERVE TISSUE.** (Ueber den  
(345) Abbau des Nervengewebes.) A. ALZHEIMER (of Munich),  
*Allg. Ztschr. f. Psych.*, Bd. 62, H. 3, 4.

THE histological methods which give useful results in the study of the brain in such organic affections as general paralysis, senile dementia, arterio-sclerotic insanity, and brain syphilis fail almost entirely when called on to aid in the histopathological differential diagnosis of the so-called functional psychoses. A few isolated tissue changes have been noticed without the subject being much elucidated: new methods are necessary, and the author suggests that the subject may be usefully attacked from a new point of view.

The few facts which we do know of the functional psychoses which lead to dementia are essentially of the nature of regressive changes. In view of this degeneration of the nerve tissue, the products of degeneration, their nature and distribution may prove to be useful indicators and helps in differential diagnosis. Marchi degeneration is of rare occurrence and of little help here. On the other hand, in the deteriorating psychoses there is a considerable

increase of fat, especially in the cells of the adventia of the blood-vessels. Other products of deterioration occur, but the methods of fixation and staining commonly employed are inadequate; new technique must be elaborated before the chemical and tinctorial qualities of these bodies can be determined, and their relations to the parenchymatous and neuroglia elements on the one hand, and to the vascular system on the other, satisfactorily worked out.

C. MACFIE CAMPBELL.

**THE PATHOLOGICAL APPEARANCES IN A CASE OF AMYOTROPHIC LATERAL SCLEROSIS, ETC.** (Un cas de sclérose latérale amyotrophique avec dégénération de la voie pyramidale suivie au Marchi de la moelle jusqu'au cortex.) ITALO ROSSI and D. ROUSSY, *Rev. Neurol.*, May 15, 1906.

THE authors describe the pathological findings in a typical case of amyotrophic lateral sclerosis. Except for the spinal ganglia, which were not obtained, the examination was very complete. The nervous tissues were examined by Marchi, Nissl, and Carmine, and the muscles by Hæmatoxylin and Eosin, and Van Gieson. They found the following lesions:—

1. Recent (Marchi) and old (Weigert) degeneration of the pyramidal system from the cord to the cortex.
2. Diffuse degeneration of the antero-lateral columns, with the exception of Gower's tract and the direct cerebellar tract.
3. Degeneration of the anterior roots in their intra-medullary course, and of the fourth and fifth dorsal roots of the left side, with corresponding ascending and descending degenerations.
4. Atrophy of the anterior horn cells of the cord.
5. Degeneration of the fibres of the hypoglossal pneumogastric and facial nerves, with cellular lesions in the nuclei.
6. Degeneration of the medullary fibres of the cortex and atrophy of the large pyramidal cells.
7. Atrophy of the peripheral nerves and muscles.

The changes in the large pyramidal cells were often slight as compared to the extent of the degeneration of the cortical fibres, which tends to show that amyotrophic lateral sclerosis is primarily of the pyramidal tracts. The degeneration of the cortical fibres supported the views of Sherrington and Campbell as regards the situation of the motor centres in the ascending frontal convolution, very few degenerated fibres being traced to the ascending parietal convolution. The changes in the cortex were limited to the large pyramidal cells and the medullary fibres, the association fibres being unaffected. No degenerated fibres were found in the corpus callosum. There was also found de-

generation in the post-longitudinal bundle in the lower part of the pons and bulb, a condition which has been previously described, and which does not alter the essentially motor character of the disease. There was sufficient degeneration in the radicular fibres of the third and sixth nerves to warrant the authors stating that they were definitely affected, but no changes were found in the corresponding fibres of the fourth nerves. Some granular bodies were found in the ventral part of the direct cerebellar tract at the level of the second and third cervical segments, but there were no changes in the cells of Clarke's column, and the authors are inclined to think that these degenerated fibres belonged to the pyramidal system rather than to the direct cerebellar tract itself. The slight degenerations which they found in the posterior columns were apparently due to the degeneration of the fourth and fifth dorsal roots of the left side. Such degenerations of the posterior roots have been described in a number of cases, and show that in amyotrophic lateral sclerosis not only may there be changes in the posterior columns, but also in the posterior nerve roots themselves. The changes in the posterior columns are analogous to those seen in cachectic states, but the rarity of the root changes makes the authors attribute them to accidental peripheral lesions rather than to a primary process analogous to that seen in the case of the motor neurone.

T. GRAINGER STEWART.

**THE INTERNAL FEATURES OF THE BRAIN OF A MICRO-  
(347) CEPHALIC IDIOT, SHOWING LACK OF THE CORPUS  
CALLOSUM.** J. F. FLASHMAN, *Reports Path. Lab. of Lunacy  
Depart., N.S.W.*, Vol. i., Part 2.

THIS brain was described in a previous part of these Reports as showing, on macroscopical examination, absence of the corpus callosum, but a study of sections stained by Weigert's method revealed the fact that this designation was incorrect, for a corpus callosum was really present, though in a very imperfect state.

Besides this, there were apparently two striking abnormalities in the brain visible to the naked eye. The one was a large bulging mass in the anterior portion of the base of the brain, which microscopical examination showed to be formed by the fusion of the heads of the two caudate nuclei across the middle line, and covered only by a thin layer of cortex. The second was a large mass of grey matter situated mesially between the hemispheres in the position of the anterior part of the corpus callosum; it consisted of a superficial layer of normal cortical tissue, and a deeper portion formed by the more or less complete fusion of masses of heterotopic grey matter, containing but few medullated fibres.



Four classes of callosal fibres are described: (1) Fibres uniting the two frontal lobes which have fused across the middle line, but these can scarcely be considered as belonging to the callosal system. (2) Small bundles of fibres which cross the middle line in the mesial grey mass and are probably true commissural fibres. (3) Undoubted callosal fibres which cross behind this grey mass in a bundle which probably represents the splenium of the corpus callosum. A considerable number of these are arranged, before their decussation, in definite bundles in the dorsal wall of the lateral ventricles, thus corresponding to the tapetum of the normal brain. Passing backwards or forwards they bend into the splenium when they reach it. (4) Fibres from the neopallium traverse the floor of the lateral ventricles and decussate in the hippocampal commissure (psalterium). It is interesting that in this case the callosal and hippocampal commissures are almost continuous, there is no trace of a septum pellucidum between them.

The two lateral ventricles united across the middle line to form a single cavity, from which the third ventricle was separated only by a layer of ependyma which formed its roof. The ventral surface of the mesial grey mass was covered by ependyma; it must consequently have developed from the hemispheres. Other interesting peculiarities are the absence of decussating fibres which could correspond to the anterior commissure, and the aberrant course of a bundle of fibres which is assumed to represent the fornix. This bundle springs from the fimbria, but passes forwards along with the tænia thalami in the floor of the lateral ventricle, apparently into the anterior portion of the thalamus.

The only striking defect in the cortex was a paucity in the number of its cells.

The most important point which the description of this brain bears on is the nature and origin of the tapetum. As is well known, Onufrowicz and others have described brains in which there was no corpus callosum, yet the tapetum was normal, and it was therefore concluded that the tapetum does not contain callosal fibres. In this case the tapetal fibres could be so easily followed into the small remnant of the corpus callosum that the author believes this view is incorrect.

GORDON HOLMES.

#### THE TOXIC CAUSE IN SOME FORMS OF MENTAL DISEASE

(348) **A NEW METHOD OF INVESTIGATION.** (*La causa tossica in alcune malattie mentali. Nuovo metodo di saggio.*) REBIZZI, *Riv. di Patol. nerv. e ment.*, F. 6, 1906, p. 241.

THE author's researches are not quite finished and he hopes to publish with more detail very soon. His method consists in the

application of leeches to patients suffering from various forms of mental disease, and subsequent examination of the animals' nerve cells by Cajal's method.

After being filled with blood, each animal is allowed to live 24 hours. The experiments have been carefully controlled.

The following cases were examined: early convalescence from alcohol (2); ordinary amentia (3); pellagrous amentia (3); general paralysis (1); senile dementia (2); idiocy (5); epilepsy (5); dementia præcox (18). In the nervous system of leeches filled with the blood of normal individuals, there is a slight diffuse hypertrophy of the neurofibrils forming the reticular network. In epilepsy during a period of improvement between the convulsions the neurofibrils were normal, but during the epileptic state they were atrophied in the cell body and hypertrophied in the axis-cylinder. The same change was found in general paralysis. In senile dementia the neurofibrils were extremely atrophied. In amentia the initial hypertrophy of the neurofibrils was followed by dissolution into granules. In recovering alcoholics, idiocy, and dementia præcox, the results were negative.

The author concludes that his investigations support very strongly the view that many forms of mental disease are of toxic origin, the result of gastro-intestinal disturbance. The blood of epileptics is only toxic during the convulsive period. The toxins may act electively upon the cells of the motor area or diffusely, in which case these cells react because of increased vulnerability, the result of some congenital anomaly or alteration sustained in intrauterine life. After the convulsion, should the toxins persist, then the other centres being affected, post-epileptic confusion results.

In senile dementia and in amentia, great importance is attached to increased growth and virulence of certain germs in the intestinal canal.

In alcoholism especially is this question important, as the gastro-intestinal inflammation induced favours overgrowth and increased virulence of the organisms normally present. The mental symptoms depend upon the predominance of certain of these, and in their absence arterio-sclerosis, renal and hepatic changes may be the only result of the intestinal disturbance.

Another point insisted on is the predisposition to attack or weakness of the nervous system which results from slight meningitis or meningo-encephalitis in infancy. These infantile diseases leave areas of diminished resistance in the cortex prone to break down under toxic action. Taking this into consideration, the necessity to ascribe to organisms an elective action is diminished. The author considers dementia præcox of toxic origin, but the toxin seems to be present only in the initial phases of the disease; later on, as

in alcoholism, there follows a period in which the symptoms are the result of the brain lesion already established.

DAVID ORR.

### CLINICAL NEUROLOGY.

**OCULAR CRISES IN TABES.** (*Crises oculaires et syndromes pseudo-basedowien dans l'ataxie locomotrice.*) HASKOVEC (Soc. de Neur. de Paris), *Rev. Neurol.*, April 5, 1906.

PEL has described as ocular crises in tabes a case of that disease, in which sudden pain in the eye occurred, with lachrymation, slight exophthalmos, widening of the palpebral aperture, and ocular hypotonia. Haskovec reports a similar case in some detail. He considers the symptoms of exophthalmic goitre presented by his patient to be due entirely to the affection of the cervical sympathetic by the tabetic morbid process; a pseudo-basedowian syndrome.

S. A. K. WILSON.

**MENTAL DISORDERS IN MULTIPLE SCLEROSIS.** (*Psychische Störungen bei der multiplen Sklerose.*) RAECKE (of Kiel), *Arch. f. Psych.*, Bd. 41, H. 2.

THE author first gives a brief résumé of the views of previous writers, and shows how, with few exceptions, notably that of Müller, they recognise the large percentage of cases of multiple sclerosis which present mental symptoms. Several writers refer to a combination of multiple sclerosis and general paralysis, without stating explicitly whether typical paralytic changes in the cortex were accompanied by sclerotic foci, or whether the condition was one of very widely disseminated insular sclerosis producing a rather diffuse brain lesion. In both diseases epileptiform convulsions may occur; it is wrong to talk of a complication of multiple sclerosis with epilepsy, where the epileptiform attacks are due to the sclerotic foci. The so-called hysterical phenomena must be looked on as the direct expression of the organic disease, and not as an incidental complication.

As to the forms of mental disorder which are met with in this disease, where the mental symptoms occur in an early stage of the disease the most common forms are depressive and maniacal disorders with delirious episodes, confusion, hallucinations, and isolated delusions. The delirious episodes frequently follow epileptiform or hysteriform attacks. The depressive conditions are frequently associated with head feelings; the maniacal conditions with a silly cheerfulness and often with great irritability. Where

the mental symptoms arise at a more advanced stage of the disease, expansive delusions with the absurd exaggerations and want of judgment of the general paralytic are more common.

Among thirty-seven patients observed by the author, thirteen cases showed marked mental enfeeblement and nine cases showed other mental disorders. Only in fifteen cases was no mental defect observed, and in these the disease was not far advanced. In five cases in the initial stage of the disease there was a simple depression; in three cases there was an emotional disturbance with delirious conditions. In one case, where the disease was already far advanced, there was a characteristic paranoic condition similar to that described by other authors. Raecke reports in detail three of his cases. The first case was that of a sixteen-year-old boy, who developed attacks of dizziness, episodes of anxiety, a horrible fear of death, a few hallucinations, impulsive reactions. Hysteriform conditions, with transitory pareses and aphasia, alternated with epileptoid attacks of dizziness and sleep. From the very beginning the memory defect was prominent, and there was great instability of the emotional tone. Neither nystagmus nor intention tremor was present; the knee-jerks were normal; speech was slow, stuttering, and abrupt; the gait was staggering; writing was tremulous. Patient died about nine months after the onset of the first symptoms. Post-mortem examination confirmed the diagnosis of multiple sclerosis, there being numerous sclerotic foci in the brain and cord.

In the second case there was no microscopical examination, but the neurological symptoms made the diagnosis quite clear. Patient was a young woman twenty-one years of age, who had a first attack at the age of twelve years, with paræsthesia of the left hand. From the age of seventeen, when she had influenza with transitory paralysis, patient suffered from epileptiform attacks, which came every four to six weeks; these attacks were frequently followed by an aphasic condition, which was also present, but much less marked, in the free intervals. Fatigue made this aphasia more marked. In addition to the aphasia there was the early onset of a euphoric dementia, with later conditions of excitement and confusion.

The third patient was a man twenty-six years of age, who, two years after the onset of the disease, with pains, impaired gait, increase of reflexes, nystagmus, and speech defect, with intellectual enfeeblement and a tendency to confabulate, developed absurdly grandiose matrimonial schemes. Notwithstanding his dementia, he took considerable interest in the affairs of the ward, showed marked personal preferences, and would only discuss his delusions under favourable conditions. A cursory examination would have elicited none of his typical ideas, and this perhaps explains how such a trend may be overlooked.

C. MACFIE CAMPBELL.

**ON TWO CASES OF SUCCESSFUL OPERATION FOR TUMOUR OF  
(351) THE SPINAL MENINGES.** (Ueber zwei Fälle von erfolgreich  
operierter Rückenmarkhautgeschwulst.) OPPENHEIM and  
BORCHARDT, *Berl. klin. Wochenschr.*, June 25, 1906.

IN the first part of the paper a detailed account is given of the symptoms and course of these cases.

The first case, of a woman aged 33, began in July 1904 with pains between the shoulder-blades followed three months later by increasing weakness of the left arm and stiffness of the left leg. The paralysis of the arm was limited to the hand and finger muscles, and the left leg showed all the reflex signs of spastic paraplegia, though the right lower limb was unaffected, save for a diminution of sensitiveness to pain and temperature. Pain was felt on pressure upon the sixth and seventh left cervical transverse processes, but no changes were visible on radioscopy. The patient declined operation, and in September 1905 was much worse, the paralysis affecting also the right leg, and the bowels and bladder having become incontinent; pain, however, was diminished. The patient was then operated upon, and an intradural fibroma or fibrosarcoma 3 cm. long was found flattening the cord. Improvement began upon the day after its removal with disappearance of the spastic condition and of the Babinski sign. Two months later she was able to walk.

The second case, of a man aged 49, began in autumn of 1904 with pains in the back and legs, weakness in the right leg, and interference with the visceral reflexes. In April 1905 there was marked paralysis in the right leg, slightly increased knee-jerks, ataxia in both legs, and disturbance of all forms of sensation on both sides up to the seventh rib, together with tenderness to pressure on the fifth dorsal spine. In March 1906 an exploratory laminectomy revealed a soft dural tumour at the level of the fourth and fifth neural rings, in the middle line, and 5 cm. long. A few days after its removal, pain and spasticity were much lessened, and the urine was voided normally; two months later the patient could walk with a stick, and the reflexes and sensations had become natural.

The second part of the paper gives the surgical details of the operations, with figures, and Borchardt states the mortality of such laminectomies at 50 per cent.

JOHN D. COMRIE

**DISSEMINATED SYPHILITIC ENCEPHALITIS.** ALBERT M.  
(352) BARRETT, *Amer. Journ. of Med. Sciences*, March 1905.

THE author describes a case of syphilis admitted to Danvers Asylum, Dec. 11, 1904, six months after the initial lesion appeared.

The secondaries were well marked at this time. Physically: the left pupil was larger than right, there was partial ptosis and external strabismus of the right eye, the knee-jerks were increased, and there were coarse tremors, especially of the hands. Mentally: there was amnesia, complete disorientation, and extreme dulness. A comatose condition ensued, and the patient died Dec. 18, 1904.

Macroscopically: the pia was hazy over the convexity and base, the third nerve and left middle cerebral were imbedded in an exudate, and there was both a focal and general meningitis.

Microscopically: the vessels of the pia, and more especially the veins, showed a proliferation of the intimal endothelium, infiltration of sheath with lymphoid and plasma cells, often polynuclears, and there was more or less mural necrosis of the vessel walls. The cortex showed both diffuse and focal changes. The former was manifest in degenerative alterations of the nerve cells, neuroglial reaction and vascular proliferation with infiltration of the shorter vessels of the cortex, and extension of the meningitis. Nissl's rod cells were everywhere numerous. The focal changes in the cortex consisted of small areas of granulation tissue, anæmic necrosis and punctate hæmorrhages, and conditions resembling small, gummata. The changes in the oblongata and cord were similar, but not so severe. Fibre degenerations were negative.

In its entirety the process is to be considered essentially as a disseminated syphilitic meningitis. Clinically the case is interesting as appearing early after syphilitic infection, and anatomically as being rather a diffuse vascular and parenchymatous condition, distinguishable from the gummatus type of syphilis on the one hand, and on the other from the cortical changes seen in general paralysis.

CHARLES I. LAMBERT.

**STATISTICAL INQUIRY INTO THE ETIOLOGY AND DURATION OF GENERAL PARALYSIS AND CAUSES OF DEATH.** (Paralisi Generale Progressiva: Etiologia—Durata—Cause di Morte.) GIUSEPPE MARGARIA, *Ann. di Fren.*, Vol. xvi., Fasc. 2.

THE material for the inquiry was provided in the records kept of the general paralytics who had died in the Turin asylum during the last ten years, amounting to 107 women and 349 men.

*Etiology.*—The disease appears to be on the increase: 49 patients died from it in the second quinquennial period, as against 42 in the first. The conditions of industrial life in large cities, with alcoholism, prostitution, and syphilis, are important factors.

Most cases of the disease occurred between the thirty-sixth and forty-fifth years. Two cases occurred between twenty-one

and twenty-five, and 4 over seventy. The proportion between male and female patients was 3·26 : 1. The age of commencement of the disease was the same for both sexes.

56·5 of the patients belonged to the poor classes, and 43·5 to the well-to-do.

83·8 came from the cities, and 16·2 from the country.

Of 349 male patients, only 13 belonged to the army.

Alcohol was found as a sole cause in 127 cases—109 men and 18 women, or in 27·87 per cent.; and syphilis in 11·46 per cent. of the men and 11·21 per cent. of the women.

Cases in which syphilis was associated with alcoholism amounted to 9·43 per cent. for the men and 4·67 per cent. for the women. Syphilis was thus found in 19·77 per cent. of all the cases examined.

*Duration.*—When the cause was alcoholism, death occurred most frequently between the thirteenth and the eighteenth months, and the same result was found in the case of syphilis.

The course of the disease depends principally on the degree of intensity of the intoxication, the association of other causes, and especially the power of individual resistance.

*Causes of Death.*—Marasmus is more common in men than in women—42·12 per cent. to 28·03 per cent. Septicæmia—due to bed-sores—on the other hand, is more common in women—23·36 per cent. to 8·59 per cent. Apoplectiform seizures are slightly more common in women than in men—12·14 per cent. to 11·74 per cent. The reverse holds for epileptiform seizures—men, 6·30 per cent.; women, 4·67 per cent.

T. C. MACKENZIE.

**SYMPTOMS OF FRONTAL DISEASE.** (*Symptome der Stirnhirnerkrankungen.*) ANTON, *Münch. med. Wchnschr.*, July 3, 1906, S. 1289.

WITH a paired organ like the frontal (pre-frontal) region, abundantly connected with another paired organ, the cerebellum, considerable compensation can occur, and the results of injury or disease may only be recognisable through a quantitative diminution in activity.

The chief physical signs are disturbance of balance in standing and walking, almost exactly as in cerebellar disease; change in character of gait (hypotonus), and loss of fine co-ordination of movement in the upper extremities. From the proximity of the motor cortex, pareses and motor aphasia are frequent complications, and in localised orbito-frontal cases, anosmia.

Abscess, etc., may be present for years unrecognised. In one-

sided disease, characteristic psychical symptoms are not evident, but from involvement of both sides there results a set of symptoms closely resembling those of general paralysis.

J. H. HARVEY PIRIE.

**SIGNIFICANCE OF JACKSONIAN EPILEPSY IN TOPICAL (355) CEREBRAL DIAGNOSIS.** (Ueber die Bedeutung der Jackson'schen Epilepsie für die topische Hirndiagnostik.) K. BONHOEFFER, *Berl. klin. Wchnschr.*, July 9, 1906, S. 935.

THE author summarises his past ten years' experience of Jacksonian epilepsy. It is a frequent symptom of organic disease of the central convolutions. In cases of traumatic hæmorrhage it is of great importance as indicating the site for trephining, but several cases of attacks in alcoholic subjects are given where the main lesion was found not to be over the motor cortex, but in some other part of the same hemisphere.

The differential diagnosis of Jacksonian epilepsy from those cases of genuine epilepsy affecting mainly or entirely one side of the body, lies chiefly in the different anamnesis, and in the complete loss of consciousness, usually, though not invariably, present in the latter. Midway between those two lie genuine epileptic cases where some other local lesion determines the one-sided character of the seizures. Here consciousness is generally lost, but a case is recorded with cerebellar atrophy on the same side as the fits where this was not the case.

A hemistatus epilepticus is frequently associated with other signs of cerebral abscess or tumour which may be present. A very interesting case is given where abscess was diagnosed, but both at operation and post-mortem there was found only hydrocephalus interna, with no apparent cause for the localisation of the fits—an "idiopathic" hemiepilepsy.

J. H. HARVEY PIRIE.

**ON THE PATHOLOGY OF EXOPHTHALMIC GOITRE.** (Zur (356) Pathologie der Basedow'schen Krankheit.) M. BERNHARDT, *Berl. klin. Wchnschr.*, July 2, 1906, p. 905.

THE cases which the author records are, he believes, unique. The interesting point in the first case lies in the association of the characteristic symptoms and signs of exophthalmic goitre, with the presence of bilateral cervical ribs.

The second observation records the simultaneous occurrence of exophthalmic goitre in husband and wife.

W. T. RITCHIE.



**THE THALAMIC SYNDROME.** (*Le syndrome thalamique.*) DE-  
(357) JÉRINE and ROUSSY, *Rev. Neurol.*, June 30, 1906, p. 521.

THIS paper is based on clinical research, supplemented by pathological investigation, and it contains a lucid and concise statement of important facts.

The onset of the hemiplegia is usually insidious: there is seldom, if ever, an actual "stroke," or any loss of consciousness. Voluntary movements on the affected side are relatively well preserved, but post-hemiplegic movements, such as hemichorea and hemiathetosis, are frequent. There is never any hemitremor, but nearly always a certain degree of hemiataxia. This is revealed in hesitation and accompanying slight inaccuracy of co-ordination, but the desired end is always attained. Apparently the ataxia is not dependent on sensory defect.

In every instance Babinski's sign is absent; that is to say, the normal flexor response is obtained. The other cutaneous reflexes are normal or absent.

Disturbances of sensation are of prime importance. Objective changes are met with, involving touch, pain, and temperature senses; the anæsthesia is never absolute, and is more marked peripherally than centrally. There is usually some failure to appreciate the niceties of the localisation of touch, delay in recognising stimuli, some atopognosis, and some widening of Weber's circles.

Deep sensation is more definitely affected: there is complete loss of the muscular sense, and sometimes diminution or loss of pallæsthesia. Astereognosis is frequent, and the sense of weight and of resistance is equally at fault.

Subjective alterations are very significant; we meet with paroxysmal pains on the affected side, referred usually to the skin or to tissues just below it, and radiating throughout the limbs. Sometimes any touch suffices to evoke them. Associated with the pains are various paræsthesiæ: occasionally there is hyperæsthesia of the hemiplegic side, sometimes an "anæsthesia dolorosa." Sometimes the pain prevents the patient from moving his limbs, suggesting a resemblance to what is known as "akinesia algera."

Sphincter troubles are not unknown. Vasomotor, trophic, and secretory disturbances are fairly frequent. Hemianopia may occur if the posterior and inferior part of the optic thalamus be involved in the lesion.

Pathological evidence is offered by the authors to show that the clinical picture which has been sketched is associated with destruction or alteration in the postero-external division of the external nucleus, together with part of the middle and internal nuclei and the corresponding fragment of the internal capsule. The motor defect is in proportion to the extent of the capsular

lesion, not of the thalamic. Experiments on apes make it clear that a local lesion in the thalamus does not affect motility. The sensory disturbances are obviously the result of thalamic involvement.  
S. A. K. WILSON.

**REVISION OF THE QUESTION OF APHASIA. THE THIRD (358) LEFT FRONTAL CONVOLUTION DOES NOT PLAY ANY SPECIAL RÔLE IN THE FUNCTION OF SPEECH.** (Revision de la question de l'aphasie. La troisième circonvolution frontale ne joue aucun rôle spécial dans la fonction du langage.)  
PIERRE MARIE, *La semaine médicale*, May 23, 1906, p. 241.

THE results, however startling they may appear, to which Professor Marie feels himself committed, have been obtained by the clinical and pathological examination of fifty cases of aphasia which have occurred in his *service* at Bicêtre.

Marie's first generalisation is, that in *every* case of aphasia there exists more or less pronounced inability to comprehend spoken language. An aphasic, however slight be his failing, can never execute correctly any complicated request. Yet this does not arise from any word deafness, because if the question be asked clause by clause, he understands and obeys accurately enough. It follows that there must be diminution in his intellectual capacity. According to Marie, sensory aphasia (Wernicke's) is not the consequence of destruction of the sensory images of language, because he does not believe in their existence. Diminution of intelligence is at the foundation of this form of aphasia. He supports his contention by a consideration of the question of mimicry. The aphasic's descriptive mimicry, by gesture, is often very feeble. Further, there is commonly diminution in the stock of things learnt by didactic processes. One of his cases was a chef who made gross mistakes in the cooking of an omelette. Superficial examination of cases of aphasia might lead one to suppose that sometimes the intellect is unimpaired, and this view is supported by the fact that in aphasics there is exaggeration of the affective reactions. Nevertheless, careful interrogation and investigation will, Marie believes, make it clear that defect of intellectual powers is fundamental.

He is not desirous of straining the argumentative value of cases in which a lesion of the third left frontal convolution was unaccompanied by motor aphasia, or cases in which that area of the cortex was unaffected, yet Broca's aphasia was clinically manifest. He agrees, of course, that the two clinical types, motor aphasia and sensory aphasia, exist; but he considers the sole essential difference to be that Broca's aphasic cannot speak, while Wernicke's aphasic can. Both are incapable of understanding complicated

questions. He quotes cases of Broca's aphasia in which word deafness was present, associated with alexia and agraphia (Thomas and Roux).

His second generalisation, therefore, is that motor aphasia is sensory aphasia plus anarthria, which anarthria is due to a lesion of the neighbouring lenticular nucleus. The sole territory, a lesion of which will produce aphasia, is comprised in the supramarginal, angular, and upper two temporal convolutions on the left side. Any lesion in this area will cause aphasia: the degree of the aphasia will depend on the extent of the lesion. This is merely a specific instance of a synthetic dogma to which Marie assigns great importance: the global production of cerebral hemisyndromes by the lesion of only a portion of the zone from which they arise. Another instance may be quoted, viz. his belief that there is no localisation in the internal capsule, and that a slight limited lesion there produces a slight hemiplegia in face, arm, and leg together. The degree of hemiplegia depends on the extent of the lesion.

As a result of his study of pathological material, Marie concludes, and gives anatomical reasons for his conclusions, that—

1. If the lesion be anterior to a transverse line passing through the fissure separating the third frontal convolution from the insula, there is neither aphasia nor anarthria.

2. If the lesion be posterior to this line, and bounded by another passing through the posterior part of the insula and the posterior extremity of the lenticular nucleus, there is anarthria.

3. If the lesion be posterior to this second line, there is aphasia.

If there be, clinically, Broca's aphasia from a lesion of the third frontal convolution, it is because the lesion is not strictly confined to the cortex, but involves the isthmus of white matter between it and the lenticular nucleus.

S. A. K. WILSON.

#### **SENSORY APHASIA: ITS LOCALISATION AND PATHOLOGICAL**

(359) **PHYSIOLOGY.** (*L'aphasie sensorielle : sa localisation et sa physiologie pathologique.*) DÉJÉRINE, *La presse médicale*, July 1906, p. 453.

THIS brief contribution is in part an answer to some of the statements recently made by Pierre Marie, which necessitate a reconsideration of the question of aphasia.

Déjérine gives a succinct historical résumé of Wernicke's sensory aphasia, mentioning such men as Bouillaud, Broca, Bastian, Wernicke, by whose labours the present doctrine has been established. It is possible that all have been mistaken, but the hypothesis is rather overwhelming, although Déjérine admires the tone of conviction with which Marie has written his article.

He proceeds to express his astonishment at the way in which Marie ignores the whole groundwork of the images of language on which modern theories are based. For Marie they do not exist; yet every physiologist and every psychologist is cognisant of them. Déjérine regrets that Marie ignores many well-differentiated forms of aphasia, subcortical and otherwise, and complains that to attribute so much to mere "intellectual impairment" is quite inadequate, in view of the point our knowledge of cortical processes has reached. He emphasises the great and fundamental difference between word deafness and mental deterioration, and quotes cases, in his turn, in which not the slightest psychical defect was discoverable. Disorders of speech are far too specialised to be explained by such generalisations as Marie offers. "Mental defect" of the type described by Marie occurs only when the area affected is that which contains the images of language, and the significance of this fact is so obvious that Déjérine is content to let the matter end thus.

S. A. K. WILSON.

**A CLINICAL AND PATHOLOGICAL CONTRIBUTION TO THE  
(360) STUDY OF APHASIA.** (Contributo clinico ed anatomo-  
patologico allo studio dell' afasia.) R. BONFIGLI, *Riv. di*  
*Patol. nerv. e ment.*, 1906, p. 266.

THE patient was a chronic alcoholic who, after a cerebral hæmorrhage, was admitted to the asylum at Rome with partial word-deafness, severe par-aphasic errors in repeating words heard, also word-blindness, writing from a copy being impossible and spontaneous writing being full of par-agraphic errors, the latter being somewhat less numerous when writing from dictation. The autopsy showed two old areas of softening in the left cerebral hemisphere, the one implicating the cortex and white matter of the angular gyrus, the other in the posterior fourth of the middle temporal convolution. The immediate cause of death was a recent hæmorrhage into the lateral ventricle.

The writer discusses the connection between the clinical symptoms and the appearances found after death. He agrees that the explanation of the word- and object-blindness, in confirmation of Bastian's views, is probably to be found in a destruction of the occipito-temporal commissure between the visual centre and the auditory centre, this commissure being interrupted by the lesion in the angular gyrus. In this particular case the patient could not be said to have what Dejerine has called the "servile" variety of writing, inasmuch as even when copying the patient made "par-agraphic" mistakes, and the power of writing to dictation was not entirely lost. He therefore had merely an attenuated form of

"servile" writing. Bonfigli thinks it probable that there are individual differences in different people as regards the cortical mechanism of words in writing and in reading. In this particular patient the power of writing to dictation was almost lost. The greater part of the word-hearing centre having remained intact, the patient could still write correctly an occasional word to dictation, whilst in the act of copying he failed completely, being unable to bring into action the word-seeing centre which in him was of minor functional importance. It is also of importance to bear in mind that there are individual differences in the extent to which the other half of the cerebrum can compensate for a focal lesion of the speech centres.

PURVES STEWART.

**ON AGRAMMATISMUS AND DISTURBANCE OF INTERNAL (361) LANGUAGE.** (*Ueber Agrammatismus und die Störung der inneren Sprache.*) K. HEILBRONNER (of Utrecht), *Arch. f. Psych.*, Bd. 41, H. 2.

PATIENT was a young man, aged 19, who one year before admission received a punctured wound in the left temporal region, followed by a severe hæmorrhage and unconsciousness lasting two weeks. He was speechless for four weeks, after which he again began to speak, but with difficulty. For three months there was complete right-sided paralysis, then some return of movement on that side. The patient remained in the clinic from June 1902 to September 1903. During the whole of this period patient had an interesting speech disorder, which showed no change. The author gives the record of one complete examination of his speech disorder, and then discusses the symptoms in detail.

Patient showed no difficulty whatever in understanding spoken questions and demands. He was able to understand written and printed matter, although he complained that it took him longer to read the newspaper, and was a little more difficult than previously. Patient spoke with some difficulty, and sounds were produced only after a considerable latent period. Patient referred to this as a mechanical difficulty. The most interesting point in his speech was the presence of Agrammatismus; the French distinguish two varieties—"negro style with verbs in the infinitive," and "telegraphic style, the phrase being reduced to the elements necessary for comprehension." In the present case both these forms were present; notwithstanding the faulty grammatical formation the sentences were almost always intelligible. In discussing the symptom, Heilbronner first excludes the explanation that, owing to the difficulty of expression, only the most important words were brought forth, thus producing a skeleton of normal speech.

He refuses this explanation for three reasons: because there was the same difficulty in writing; because the difficulty was not merely a difficulty of production, as was shown by the fact that the patient made errors even in the simple task of giving nouns their correct article; finally because, when given a few words, he was unable to compose sentences out of them; he understood the task, was displeased with his results, but could not produce a satisfactory result.

Ziehen considers a general intellectual disorder as a necessary condition of Agrammatismus, and holds that this symptom in an aphasic means a co-existing mental enfeeblement. In this case there was no foundation for such an assumption, and Heilbronner looks upon the Agrammatismus as a symptom of a focal disorder. He passes on to discuss the anatomical relations of such a lesion. Sensory aphasics never show the symptom, and in the disappearance of a sensory aphasia a stage with this symptom has not been observed, whereas it has been observed during improvement after motor aphasia. In this context the author refers to a previously published case of transcortical motor aphasia; the patient, like the present one, understood speech, could repeat spoken sentences, but when using the ordinary phrases at his command only rarely uttered concrete words, while the present patient used these latter freely, but did not join them together with the usual grammatical parts of speech. The former patient showed the maximum disorder of the expressive part of the speech mechanism which is possible without implication of Broca's area: in the present case the lesion must be still nearer to the motor centre, and probably implicates this latter; the implication may be deduced from the difficulty of expression. A lesion, therefore, in the motor territory, even leading to Agrammatismus and disorder of internal language, need not influence the power to find words. In this case, as in his earlier case, Heilbronner does not consider the symptom as a stage in the regression of a motor aphasia, but rather as an independent focal symptom, and he holds that the other symptoms leading to the complete transitory aphasia were merely irritative and owing to temporary disturbance. The author next passes to the discussion of certain symptoms which are usually regarded as an expression of disturbance of "the internal language." Owing to the general mental condition of patient, this disorder was particularly easy to define. Patient showed a loss of grasp of the internal structure of words quite apart from their meaning. While able to choose the suitable word to express his ideas, and while able to complete words of which he was given only the initial meaningless syllable, he was unable to grasp the structure of words with regard to their letter components. He could pronounce the individual letters, could pick out letters named and write down

letters demanded, but he was unable, if given the component letters of a word, to put the word together, and could not spell even simple words correctly. The fact that the erroneous products on different occasions showed striking similarity points to the importance of studying not only the disordered function, but also the incorrect products, to see what are the laws which govern them. This disorder of the internal language was in striking contrast with the almost complete absence of paraphasia in the spontaneous speech of the patient, and with the very slight degree of paraphasia which was seen in the spontaneous writing of the patient. In conclusion the author touches lightly the question of the importance of the motor speech centre for the understanding of speech, and in this context he emphasises the fact of individual variation.

The author sums up as follows: 1. Agrammatismus can follow a trifling motor speech disorder. 2. It may remain stationary for years, even under favourable conditions. 3. Agrammatismus in an aphasic is not necessarily associated with mental enfeeblement. 4. Agrammatismus in motor aphasia is a primary defect phenomenon. 5. Well-marked Agrammatismus may co-exist with practically intact understanding of connected discourse. 6. The sequels of a light motor speech disturbance can affect more severely the structure of sentences than the structure of words. 7. As in the case of Agrammatismus, so with regard to the structure of words—the disorder is more marked on the expressive side (writing) than on the receptive side (reading.) 8. The finding of words may remain intact notwithstanding Agrammatismus and the disturbance of the grasp of the internal structure of words. 9. The laws which determine the nature of the errors made form a suitable subject for future study.

C. MACFIE CAMPBELL.

**HYSTERICAL DYSARTHRIA.** (*Dysarthrie Hystérique.*) DEBOVE, (362) *Arch. Gén. de Méd.*, July 10, 1906, p. 1746.

THE source of this article was a clinical lecture given in respect to a male patient, aged thirty-two. The characters of his speech were as follows: The accent was Alsatian, and b's and d's were replaced by p's and t's. His speech was voluble and nasal, and could be understood only when delivered slowly. He repeats words more distinctly than he speaks them spontaneously. There was no difficulty in naming objects or in writing. He sings and reads music well; he also draws very well. There were traces of a right hemiparesis in face, arm, and leg. Still he could thread a needle and sew with the right hand. The deep reflexes were slightly exaggerated, but were equal on the two sides. There

were no sensory troubles, but lividity of the hands showed that some vaso-motor defect was present. At times, under the influence of emotion or of a sudden startling, crises of general trembling appeared.

The case was then one of dysarthria, of articulatory defect, and not of aphasia. The decision as to whether that is functional or organic is not so easily arrived at. A bulbar lesion may be excluded, for there is no motor trouble with the tongue, lips, palate, or larynx. There was no physical sign of hysteria beyond the mental changes; no anæsthesia, hysterogenetic zones, or contraction of the visual field. The patient was very suggestionable, improvement being obtained by appropriate affirmation. The feature most indicative of hysteria was the description written by the patient of three attacks: it was after the last of these that he was left with the dysarthria and hemiplegia. The attacks consisted of trembling, oppression, and several distinctly lyssophobic symptoms.

ERNEST JONES.

**THE IMPORTANCE OF LUMBAR PUNCTURE IN PSYCHIATRY (363) AND NEUROLOGY.** (Sull' importanza della puntura lombare nella Psichiatria e Nevrologia.) L. MERZBACHER, *Riv. di Patol. nerv. e ment.*, 1906, p. 193.

THE writer gives an account of the ordinary technique of lumbar puncture as practised at the Heidelberg clinic. At the outset of his paper he expresses his surprise that lumbar puncture has not yet been introduced amongst diagnostic methods in the Italian clinics of neurology and psychiatry, and laments his failure to find any references to the subject in Italian medical literature. Merzbacher's self-imposed mission of scientific evangelist will doubtless amuse the Italian neurologists, to whom lumbar puncture has been familiar both in theory and in practice for some years, as evidenced by the numerous valuable contributions on the subject by Italian authors, amongst which we might mention an article by Pighini (*Rivista di Freniatria*, 1903, p. 381), which contains practically all the facts to which Merzbacher now draws attention, and many more.

The paper is a fair elementary account of lumbar puncture. It re-states a number of familiar facts already established by neurologists working in the outer darkness of France, of England, of Italy, and of Germany (outside Heidelberg). Three of the four illustrations are copied (without acknowledgment) from an article by Desfosses and Dumont five years ago (*La Presse Médicale*, 1901, p. 268). The remaining figure, however, a careful study of three glass tubes and two pipettes, is apparently original.



His chief conclusions are as follows:—Every case of general paralysis and of tabes shows marked lymphocytosis of the cerebro-spinal fluid. In general paralysis there is an excessive amount of albumin in the fluid; in the case of tabes there is no excess. The occurrence of syphilis, even without signs or symptoms of disease of the central nervous system, produces in nine-tenths of cases a remarkable increase in the number of lymphocytes, the albumin not being increased. (This statement is not in agreement with the observations of certain other writers. Thus the reviewer,<sup>1</sup> in a series of twelve syphilitic cases at various stages of the disease, found that the increase of lymphocytes was so small as to be practically negligible.) All forms of meningitis are accompanied by marked lymphocytosis. There is no form of psychosis in non-syphilitic patients which is accompanied by lymphocytosis of the cerebro-spinal fluid. Lymphocytosis is the earliest and most constant sign of tabes and of general paralysis of the insane.

PURVES STEWART.

### PSYCHIATRY.

**THE POSITION OF THE ATYPICAL CHILD.** W. H. GROSZMANN,  
(364) *Journ. of Nerv. and Ment. Dis.*, July 1906.

THE public and private schools of to-day are overlooking the educational needs of a very appreciable fraction of school children. Educators recognise only the patent cases of idiocy, imbecility, epilepsy, etc., to the exclusion of about ten per cent. of the total number of children who may be classed as "non-typical," or below the "essential average." The task of introducing methods applicable to the child whose mental, moral, and physical condition is warped, has not been sufficiently investigated.

The "non-typical" children are classified as follows:—(1) Congenitally abnormal children; (2) Congenitally defective children; (3) Children of rudimentary development; (4) Children of arrested development; (5) Atypical children proper; (6) Pseudo-atypical children; (7) "Average," "ordinary," "typical" children.

An atypical child is an embodiment of numerous warped tendencies which continue to deviate from the average to the abnormal, with increase in age.

The home environment is the direct abettor of the child's peculiar growth, the parents themselves are frequently abnormal and they fail to correct their children because their own attitude is irrational. The beneficial effect which the ordinary school has upon many children is wholly inadequate to overbalance the effects of home training.

<sup>1</sup> *Edinburgh Medical Journal*, 1906, p. 429.

The private tutor is also a failure in such a home, because his methods must be modified to comply with the distorted views of the child's parents. Such children must be removed into an environment where perfect harmony and interaction exist between all influences; rigorous hygienic treatment and the co-operation of a medical specialist are invaluable. State or municipal institutions are necessary to obtain this end; private endeavours can only indicate the path. From an economical standpoint the results will warrant the expense, for the atypical child, changed into a normal man or woman, is a valuable aid to our social structure. From an educational standpoint nothing can give more promise.

C. H. HOLMES.

---

## Review

**PSYCHOTHERAPEUTISCHE BRIEFE.** Prof. Dr H. OPPENHEIM.  
S. Karger, Karlstrasse 15, Berlin, 1906, M. 1.

THIS is a brochure of forty-four pages in the form of letters supposed to have been written by Professor Oppenheim to patients under his care. The letters are really expansions of others actually written, and they also incorporate advice given orally at consultations to patients suffering from various forms of functional nervous disease, which may be included shortly under the terms of neurasthenia or psychasthenia. The letters are eleven in number, and each one deals with the treatment of a special variety of functional nervous disease. They are intended as a guide to young physicians entering on practice as to how to deal with a very difficult class of disease, but they deserve to be read and pondered over by old as well as young. They are written in a quiet, dignified, impressive style, and each one unveils the course of treatment that has been adopted, and shows the form of reasoned suggestion or other method which seems to be most suitable in each case.

The following are some of the subjects dealt with: a settled but unfounded conviction of organic brain disease; an eye disease of psychogenic origin; insomnia and its aggravation by the dread of the effects of sleeplessness; a dread of the harmful effects of effort; mental depression and incapacity resulting from an unsuitable marriage; the fears which arise from the discovery of the existence of an incipient but not necessarily progressive disease of the spinal cord; the dread of failure of mental faculty in an artist; vertigo in an aged general, who has become entirely

unmanned by the unfounded dread of calcification of the cerebral arteries; the insufferable egotism of a neurasthenic lady, whose family had been made to dance attendance upon her during four years' rest in bed.

The advice given is so sound and so detailed that it may be hoped that the letters will be accessible at no distant date to English and American physicians in their own language.

ALEXANDER BRUCE.

## Bibliography

### ANATOMY

- M. et MME. DEJERINE. Les Colonnes Cellulaires des Cornes Antérieures de la Moelle épinière de l'homme. (Soc. de Neurol.) *Rev. Neurol.*, juillet 30, 1906, p. 689.
- SCHIEFFERDECKER. Neurone und Neuronenbahnen. Barth, Leipzig, 1906, M. 11.
- QUENSEL. Beitrag zur Kenntnis der Grosshirnfaserung. *Monatssch. f. Psychiat. u. Neurol.*, Aug. 1906, S. 166.
- CARMELO CIACCIO. Sulla fina struttura degli elementi del simpatico periferico. *Ann. di Neurologia*, Anno xxiv., f. 2-3, 1906, p. 159.
- BONNEY. Eine neue und leicht auszuführende dreifache Färbung für Zellen und Gewebeschnitte nach Flemmings Dreifachbehandlung. *Virchows Arch.*, Bd. 185, H. 2, 1906, S. 359.
- VAN DER VLOET. Ueber den Verlauf der Pyramidenbahn bei niederem Säugetieren. *Anat. Anzeiger*, Aug. 3, 1906, S. 113.
- MABEL FITZGERALD. An Investigation into the Structure of the Lumbosacral-coccygeal Cord of the Macaque Monkey. *Proc. of the Roy. Soc.*, Vol. B. 78, No. B. 523, 1906, p. 88.
- ERIK WARFROMGÉ. Beitrag zur Kenntnis der spinalen und sympathischen Ganglienzellen des Frosches. *Arch. f. mikros. Anat.*, Bd. 68, H. 3, 1906, S. 432.
- G. E. COGHILL. The Cranial Nerves of Triton Taeniatus. *Journ. Comp. Neurol. and Psychol.*, July 1906, p. 247.

### PHYSIOLOGY

- PROBST. Über die zentralen Sinnesbahnen und die Sinneszentren des menschlichen Gehirnes. Holder, Wien, 1906, M. 3.45.
- MAX VERWORM. Die Vorgänge in den Elementen des Nervensystems. *Ztschr. f. allg. Physiol.*, Bd. 6, H. 2, 1906, S. 11.
- Z. TRÉVES. Bemerkungen zum Aufsatz: O. Zoth: Ergographische Versuche in die Erholung des Muskels. *Arch. f. die ges. Physiol.*, Bd. 113, H. 9-10, 1906, S. 529.
- SCHUSTER. Untersuchungen über die Sensibilitätsleitung im Rückenmark des Hundes. *Monatssch. f. Psychiat. u. Neurol.*, Aug. 1906, S. 97.
- KEITH LUCAS. On the Optimal Electric Stimuli of Normal and Curarised Muscle. *Journ. Physiol.*, Aug. 10, 1906, p. 372.
- PATRIZI et FRANCHINI. Quelques particularités sur l'arrêt de la respiration par stimulation centripète du vague. Fatigue et restauration du réflex inhibiteur respiratoire. *Arch. ital. de Biol.*, Vol. xlv., f. 3, 1906, p. 416.
- FINGONI et PEA. Sur le centre et les nerfs sécréteurs du rein. *Arch. ital. de Biol.*, Vol. xlv., f. 3, 1906, p. 369.
- CHIARMI. Changements morphologiques qui se produisent dans la rétine des vertébrés par l'action de la lumière et de l'obscurité. *Arch. ital. de Biol.*, Vol. xlv., f. 3, 1906, p. 337.
- JAMES CARLETON BELL. The Reactions of Crayfish to Chemical Stimuli. *Journ. Comp. Neurol. and Psychol.*, July 1906, p. 299.

## PSYCHOLOGY

- PIERON. Le XIV<sup>e</sup> Congrès Annuel de l'American Psychological Association. *Rev. de Psychiat.*, juillet 1906, p. 185.
- MAX VERWORN. Die cellularphysiologische Grundlage des Gedächtnisses. *Ztschr. f. allg. Physiol.*, Bd. 6, H. 2, 1906, S. 119.
- MÜLLER. Le problème du grossissement des astres au point de vue méthodologique. *Arch. de Psychol.*, T. v., No. 20, 1906, p. 305.
- NUËL. La psychologie comparée est-elle légitime? *Arch. de Psychol.*, T. v., No. 20, 1906, p. 328.
- BENTLEY. The Psychology of Organic Movements. *Amer. Journ. Psychol.*, July 1906, p. 293.
- HAYES. A Study of the Affective Qualities. *Amer. Journ. Psychol.*, July 1906, p. 353.
- GESELL. Accuracy in Handwriting as related to School Intelligence and Sex. *Amer. Journ. Psychol.*, July 1906, p. 394.
- FOSTER and GAMBLE. The Effect of Music on Thoracic Breathing. *Amer. Journ. Psychol.*, July 1906, p. 406.
- LEON ASHER. Das Gesetz der spezifischen Sinnesenergie und seine Beziehung zur Entwicklungslehre. *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. 41, H. 3, 1906, S. 157.
- HANS RÜPP. Über Lokalisation von Druckreizen der Hände bei verschiedenen Lagen der letzteren. (Schluss.) *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. 41, H. 3, 1906, S. 182.
- HEYMANS und WIERSMA. Beiträge zur speziellen Psychologie auf Grund der Massenuntersuchung. (Schluss.) *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. 42, H. 4 u. 5, 1906, S. 258.
- PICK. Rückwirkung sprachlicher Perseveration auf den Assoziationsvorgang. *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. 42, H. 4 u. 5, 1906, S. 241.
- KATZ. Experimentelle Beitrag zur Psychologie des Vergleichs im Gebiete des Zeitsinns. *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. 42, H. 4 u. 5, 1906, S. 302.
- PORTER. The Habits, Instincts, and Mental Powers of Spiders, Genera *Aigrope* and *Epeira*. *Amer. Journ. Psychol.*, July 1906, p. 306.

## PATHOLOGY

- S. WALTER RANSON. Retrograde Degeneration in the Spinal Nerves. *Journ. Comp. Neurol. and Psychol.*, July 1906, p. 265.
- GEORG EISATH. Ueber normale und pathologische Histologie der menschlichen Neuroglia. *Monatsschr. f. Psychiat. u. Neurol.*, Aug. 1906, S. 139.
- BASIL KILVINGTON and W. A. OSBORNE. The Regeneration of Post-Ganglionic Vaso-Constrictor Nerves. *Journ. Physiol.*, Aug. 10, 1906, p. 267.
- LUGARO. Ancora un' esperienza contro l' autorigenrazione delle fibre nervose. *Riv. di Patol. nerv. e ment.*, Vol. xi, f. 6, 1906, p. 273.
- PERUSINI. Ueber die Veränderungen des Achsenzylinders und der Marksheide im Rückenmark bei der Formalfixierung. *Ztschr. f. Heilk.*, Bd. 27, H. 7, 1906, S. 193.
- HARVEY BAIRD. The Pathology of Four Cases of Epileptic Idiocy. *Journ. Ment. Sc.*, July 1906, p. 571.
- LEWIS BRUCE. A Serum Reaction occurring in Persons suffering from Infective Conditions. *Journ. Ment. Sc.*, July 1906, p. 515.
- LIEBSCHER. Zur Kenntnis der Mikrogyrie nebst einigen Bemerkungen und die sogenannten Heterotopien um Rückenmarke des Menschen. *Ztschr. f. Heilk.*, Bd. 27, H. 7, 1906, S. 219.
- HANS EVENSEN. The Pathology of General Paralysis. *Rev. Neurol. and Psychiat.*, Aug. 1906, p. 537.
- ENRICO ROSSI. Nota di anatomia patologica del sistema nervoso centrale in un caso di demenza paralitica. *Ann. di Neurologia*, Anno xxiv., f. 2-3, 1906, p. 171.
- F. W. MOTT. The Microscopic Changes in the Nervous System in a Case of Chronic Dourine. *Brit. Med. Journ.*, Aug. 11, 1906, p. 300.
- LACOMME. Trypanosomes et maladie du sommeil. Schneider, Lyon, 1906.
- KRASTING. Beitrag zur Statistik und Kasuistik metastatischen Tumoren, besonders der Carcinommetastasen im Zentralnervensystem. *Ztschr. f. Krebsforschung*, Bd. 5, H. 2, 1906, S. 315.
- HILDEBRANDT. Zur Kenntnis der gliomatösen Neubildung des Gehirns mit

- besonderer Berücksichtigung der ependymären Gliome. *Virchows Arch.*, Bd. 185, H. 2, 1906, S. 341.
- D'ABUNDO. Experimentell erzeugte Gehirn-Atrophie und damit verbundene Schädel-Atrophie. *Centralbl. f. Nervenheilk. u. Psychiat.*, Aug. 15, 1906, S. 625.
- HERBERT E. ROAF and C. S. SHERRINGTON. Experiments in Examination of the "Locked-jaw" induced by Tetanus Toxin. *Journ. Physiol.*, Aug. 10, 1906, p. 315.
- G. D'ABUNDO. Patologia sperimentale spinale. *Ann. di Neurologia*, Anno xxiv., f. 2-3, 1906, p. 149.

## CLINICAL NEUROLOGY AND PSYCHIATRY

### GENERAL—

- PEARCE BAILEY. Diseases of the Nervous System resulting from Accident and Injury. Sidney Appleton, London, 1906, 21s.
- WILHELM SPECHT. The Clinical Measurements of Fatigue. I. Measurement of Mental Fatigue. *Journ. Ment. Sc.*, July 1906, p. 517.
- DOUMER. Sur l'action exercée par les rayons de Röntgen sur le système nerveux central. *Ann. d'Électrobiol. et de Radiol.*, juin 1906, p. 408.
- DUMOLARD. Recherche sur la fréquence des Maladies Nerveuses chez les indigènes musulmans d'Algérie. (Soc. de Neurol.) *Rev. Neurol.*, juillet 30, 1906, p. 697.
- W. H. B. STODDART. On Instinct: A Psycho-Physical Study in Evolution and Dissolution. *Journ. Ment. Sc.*, July 1906, p. 491.

### MUSCLES—

- LENOBLE et AUBINEAU. Une variété nouvelle de myoclonie congénitale pouvant être héréditaire et familiale à nystagmus constant. *Rev. de Méd.*, août 1906, p. 471.
- RAYMOND et LEJONNE. Deux cas de myasthénie bulbo-spinale. *Rev. Neurol.*, août 15, 1906, p. 709.
- THIELE und GRAWITZ. Ueber senile Atrophie der Augenmuskeln. *Deutsch. med. Wchenschr.*, Aug. 2, 1906, S. 1237.
- ROCHARD et de CHAMPTASSIN. Généralités sur le muscle dans ses divers états, atrophique, normal, hypertrophique. *Bull. gén. de Thérapeutique*, juillet 8, 1906, p. 5.

### PERIPHERAL NERVES—

- P. RUDAUX. Névrites gravidiques. *Arch. gén. de méd.*, juillet 31, 1906, p. 1951.
- MEDEA et ROSSI. Les résultats du Traitement Chirurgical de 17 cas de lésion traumatique des Nerfs Périphériques. (Soc. de Neurol.) *Rev. Neurol.*, juillet 30, 1906, p. 705.
- GAUSSEL et SMIRNOFF. Un cas de paralysie radiculaire du plexus brachial, type Dejerine-Klumpke, par méningite tuberculeuse rachidienne. *Rev. Neurol.*, août 15, 1906, p. 718.
- EUGENIO MEDEA. I fenomeni nevritici negli alienati e i fenomeni psicopatici nelle nevriti. *Ann. di Neurologia*, Anno xxiv., f. 2, 3, 1906, p. 113.
- ERIC MACNAMARA and JULIUS BERNSTEIN. Landry's Paralysis. *Brit. Med. Journ.*, Aug. 4, 1906, p. 248.

### SPINAL CORD—

- Tabes.**—FREDERICK W. PRICE. A Case of Tabes Dorsalis in which Widespread Cutaneous Sensory Manifestations completely disappeared. *Lancet*, July 28, 1906, p. 225.
- SCHRÖDER. Ein Beitrag zur Histopathologie der Tabes dorsalis. *Centralbl. f. Nervenheilk. u. Psychiat.*, Aug. 1, 1906, S. 585.
- Progressive Muscular Atrophy.**—VITEK. Zur Ätiologie der progressiven spinalen Muskelatrophie. *Neurol. Centralbl.*, Aug. 16, 1906, S. 753.
- HUET et LEJONNE. Un cas d'Atrophie Musculaire à type Aran-Duchenne par Poliomyélite antérieure chronique. (Soc. de Neurol.) *Rev. Neurol.*, juillet 30, 1906, p. 670.
- Amyotrophic Lateral Sclerosis.**—KOJEVNIKOFF. Atrophie non systématisée dans deux cas de Sclérose Latérale Amyotrophique. (Soc. de Neurol.) *Rev. Neurol.*, juillet 30, 1906, p. 699.

- CHARTIER et KOJEVNIKOFF. Un cas de Sclérose Latérale Amyotrophique à début douloureux et atypique. (Soc. de Neurol.) *Rev. Neurol.*, juillet 30 1906, p. 683.
- Paraplegia.**—BABINSKI. De la Paralyse per Compression du Faisceau Pyramidal, sans dégénération secondaire. (Soc. de Neurol.) *Rev. Neurol.*, juillet 30, 1906, p. 693.
- Caisson Disease.**—MACNAUGHTON. Frictional Electricity: A Factor in Caisson Disease. *Lancet*, Aug. 18, 1906, p. 435.
- Cauda equina.**—F. RAYMOND. Sur quelques affections de la Queue de Cheval. *Arch. gén. de méd.*, juillet 31, 1906, p. 1940.
- Cerebro-spinal Fluid.**—WIDAL. La lymphocytose rachidienne dans la syphilis. *Journ. des Prat.*, juillet 28, 1906, p. 468.
- TOBLER. Ueber Lymphocytose der Cerebro-spinalflüssigkeit bei kongenitaler Syphilis und ihre diagnostische Bedeutung. *Jahrb. f. Kinderheilk.*, Juli 10, 1906, S. 1.

**BRAIN—**

- Meningeal Hæmorrhage.**—APELT. Zum Kapitel der Diagnose des extra- und intraduralen traumatischen und pachymeningitischen Hämatoms. *Mitt. a. d. Grenz. d. Med. und Chir.*, Bd. 16, H. 2, 279.
- Meningitis.**—CARLOS FRANCA. Ueber zerebro-spinale Meningitis. *Woch. med. Presse*, No. 33, 1906, S. 1727.
- WESTENHOEFFER. Pathologisch-anatomische Ergebnisse der oberschlesischen Genickstarreepidemie von 1905. *Klin. Jahrb.*, Bd. 15, H. 4, 1906, S. 657.
- KIRCHNER. Die übertragbare Genickstarre in Preussen im Jahre 1905 und ihre Bekämpfung. *Klin. Jahrb.*, Bd. 15, H. 4, 1906, S. 729.
- LANNOIS et PARRETIÈRE. De la méningite otogène et de sa curabilité. *Lyon Méd.*, août 1906, p. 253.
- MAUCLAIRE. La tuberculose méningée et encéphalique. *Journ. des Prat.*, juillet 28, 1906, p. 469.
- ALBERT BLAU. Kasuistischer Beitrag zur Meningo-Encephalitis serosa. *Ztschr. f. Ohrenheilk.*, Bd. 52, H. 1-2, 1906, S. 129.
- ETIENNE RABAUD. Méningite fœtale et spina bifida. *Arch. gén. de méd.*, août 21, 1906, p. 2136.
- Encephalitis.**—WITTE. Akute Encephalitis und apoplektische Narbe des Kleinhirns. *Neurol. Centralbl.*, Aug. 16, 1906, S. 743.
- Sinus Thrombosis.**—KENNON. Symptoms and Treatment of Sinus and Jugular Thrombosis, with Report of Five Cases. *Arch. of Otol.*, June 1906, p. 189.
- Hemiplegia.**—WICHERN. Ueber zwei Fälle von zerebraler Hemiplegie im Kindesalter. *Münch. med. Wchnschr.*, Juli 31, 1906, S. 1510.
- Tumour.**—GILBERT BARLING. Removal of a Cerebral Tumour (Endothelioma) which had invaded the Overlying Cranial Bone. *Lancet*, Aug. 4, 1906, p. 282.
- G. H. GRANT DAVIE. Case of Obscure Intracranial Tumour: Meningeal Sarcoma with Extension to Fourth Ventricle. *Brit. Med. Journ.*, Aug. 11, 1906, p. 301.
- GIORDANI. Le diagnostic des tumeurs de l'hypophyse par la radiographie. *Thèse*. Baillière, Paris, 1906.
- RAYMOND CESTAN. L'épithélioma primitif du cerveau. *Gaz. des Hôp.*, août 7, 1906, p. 1059.
- Abscess.**—J. STODDART BARR. A Case of Otitic Extradural Abscess, associated with Paralysis of the Sixth Cranial Nerve and Double Optic Neuritis. *Glasgow Med. Journ.*, Aug. 1906, p. 107.
- General Paralysis.**—MARGARIA. Studio Clinico-Statistico sui morti per Paralisi Generale Progressiva. *Ann. di Freniatria*, Vol. xvi., f. 2, 1906, p. 177.
- PILTZ. Sensibilitätsstörungen bei Paralysis progressiva incipiens. *Neurol. Centralbl.*, Aug. 1, 1906, S. 690.

**MENTAL DISEASES—**

- CHARPENTIER et KHAN. Le VIe Congrès international d'Anthropologie criminelle. *Rev. de Psychiat.*, juillet 1906, p. 188.
- LEWIS C. BRUCE. Studies in Clinical Psychiatry. Macmillan & Co., London, 1906, 10s. 6d.
- PETERSSEN-BORSTEL. Gutachten über den Zusammenhang zwischen Gasvergiftung und Geisteskrankheit. *Vierteljahrs. f. gericht. Med.*, Juli 1906, S. 57.
- REBIZZI. La causa tossica in alcune malattie mentali. Nuovo metodo di saggio. *Riv. di Patol. nerv. e ment.*, Vol. xi., f. 6, 1906, p. 241.

- FERRAI. Contributo alla valutazione della imputabilità negli Stati psicopatici. *Manicomio*, Anno xxii., No. 1, 1906, p. 123.
- BOULENGER et HERMANT. Association des idées chez les idiots et les imbéciles. Gaud, Paris, 1906.
- BURZIO. Idiosia et atetosi doppia. *Ann. di Freniatria*, Vol. xvi., F. 2, 1906, p. 97.
- J. SHAW BOLTON. Amentia and Dementia. *Journ. Ment. Sc.*, July 1906, p. 221.
- TOULOUSE. Examen de quelques questions touchant à la responsabilité. *Arch. de Psychiat.*, juillet 1906, p. 165.
- HALBERCHTADT. Contribution à l'étude de la folie par contagion mentale. *Thèse.* Baillière et fils, Paris, 1906.
- TERRIEN. Phobies. *Presse méd.*, août 11, 1906, p. 497.
- FALCIOLA. L'accrescimento Ungueale nella Frenosi Maniaco-Depressiva. *Ann. di Freniatria*, Vol. xvi., f. 2, 1906, p. 117.
- LEBORGNE. Contribution à l'étude des symptômes et des lésions médullaires de la démence précoce. *Thèse.* Simon, Rennes, 1906.
- SOUKANHOFF. On Hypochondriacal Melancholia in Russian Soldiers. *Journ. Abnor. Psychol.*, Vol. i., No. 3, 1906, p. 135.
- SOUKANHOFF. Sur quelques cas particuliers de trouble mental à caractère paranoïde et mélancolique. *Rev. de Psychiat.*, juillet 1906, p. 178.
- PARHON. Un cas de mélancolie avec hypertrophie thyroïdienne succédant à la ménopause. *Rev. Neurol.*, juillet 30, 1906, p. 640.
- LEVI BIANCHINI. Observation sur les tableaux cliniques de paranoïa et démence paranoïde. *Rev. Neurol.*, juillet 30, 1906, p. 645.
- NATHAN RAW. The Mental Disorders of Pregnancy and the Puerperal Period. *Edin. Med. Journ.*, Aug. 1906, p. 118.
- ALEX. PILCZ. Beitrag zur vergleichenden Rassen Psychiatrie. Deutsche. Wien, 1906.
- URSTEIN. Ein Beitrag zur vergleichenden Psychiatrie. *Centralbl. f. Nervenh. u. Psychiat.*, Aug. 15, 1906, S. 629.
- CABANES et NASS. La Névrose révolutionnaire. *Soc. française d'impr.*, Paris, 1906, 4 fr.
- DEL GRECO. Il carattere criminale. *Manicomio*, Anno xxii., No. 1, 1906, p. 73.
- ANGIOLELLA. Genio e criminalità. *Il Manicomio*, Anno xxii., No. 1, 1906, p. 1.
- GIMBAL. Les incendiaires. (Suite et fin.) *Ann. méd.-psychol.*, juillet-août 1906, p. 32.
- ANGIOLELLA. I germi etnici e psicologici della camorra e del brigantaggio. *Manicomio*, Anno xxii., No. 1, 1906, p. 47.
- VENTRA. La Legge sui Manicomi e sugli alienati. *Manicomio*, Anno xxi., No. 1, 1906, p. 15.
- SCHNITZER. Moderne Behandlung der Geisteskranken. Walther, Berlin, 1906, M. —50.
- HOPPE. Ein Gang durch eine moderne Irrenanstalt. Marhold, Halle, 1906, M. 1.60.

#### ALCOHOL—

- BIANCHI. L'alcool e le malattie del sistema nervoso. *Ann. di Neurologia*, Anno xxiv., f. 2-3, 1906, p. 129.
- JULIUSBURGER. Ein Fall von akuter Bewusstseinsstörung alkoholischer Ätiologie. *Neurol. Centralbl.*, Aug. 16, 1906, S. 741.
- LHERMITTE et HALBERSTADT. Étude-anatomo-clinique d'un cas de psychoses de Korsakoff. *Arch. gén. de méd.*, août 14, 1906, p. 2049.
- LAUSCHNER. Zur Statistik und Pathogenese des Quinquand'schen Zeichens. *Berl. klin. Wchnschr.*, Aug. 20, 1906, S. 1124.
- W. C. SULLIVAN. Industry and Alcoholism. *Journ. Ment. Sc.*, July 1906, p. 505.

#### SPECIAL SENSES AND CRANIAL NERVES—

- STERNBERG. Subjektive Geschmacksempfindungen. *Zschr. f. Klin. Med.*, Bd. 59, H. 5 u. 6, 1906, S. 491.
- RAYMOND, LEJONNE et GALEZOWSKI. Cécité corticale par double Hémianopsie. (Soc. de Neurol.) *Rev. Neurol.*, juillet 30, 1906, p. 680.
- JEAN GALEZOWSKI. Deux cas d'Hémianopsie bitemporale. (Soc. de Neurol.) *Rev. Neurol.*, juillet 30, 1906, p. 677.
- MAGALHAES LEMOS. Perte de la vision mentale des objets (formes et couleurs) dans la mélancolie anxieuse. *Ann. méd.-psychol.*, juillet-août 1906, p. 5.

- LOHMANN. Ueber die typische Exzentricität des kleinen Istringes und das Verhältnis des Exzentricität des Sehnerven zur der Ora serrata. *Klin. Monatsbl. f. Augenheilk.*, Juli-Aug. 1906, S. 68.
- EUGEN v. HIPPEL. Ueber seltene Fälle von Lähmung der Akkommodation und von Pupillenstarre. *Klin. Monatsbl. f. Augenheilk.*, Juli-Aug. 1906, S. 97.
- TERSON. La Paralysie du Moteur oculaire externe au cours des Otites. *Soc. franç. d'ophtal.*, mai 10, 1906.
- STEINERT und BIELSCHOWSKY. Ein Beitrag zur Physiologie und Pathologie der vertikalen Blickbewegung. *Münch. med. Wchnschr.*, Aug. 14, 1906, S. 1613.
- TÖDTER. Ein Beitrag zur isolierten Blicklähmung nach oben und unten. *Klin. Monatsbl. f. Augenheilk.*, Juli-Aug. 1906, S. 102.
- BARARANY. Untersuchungen über den vom Vestibularapparat des Ohres reflektorisch ausgelösten rhythmischen Nystagmus und seine Begleiterscheinungen. Coblenz, Berlin, 1906, M. 2.50.
- GEORGE F. STILL. A Lecture on Head-Nodding with Nystagmus in Infancy. *Lancet*, July 28, 1906, p. 207.
- VAN DER VLOET. Ueber die Ursache des Eintrittes der Entartungssteigerung in den Muskeln der gelähmten Gesichtshälfte. *Monatsschr. f. Psychiat. u. Neurol.*, Aug. 1906, S. 188.
- ALLAIRE. Sur deux cas de paralysie faciale protubérantielle. *Bull. de la Soc. Française d'Électrothér.*, août-sept. 1906, p. 185.
- MIRALLIÉ. Paralysie faciale périphérique Autopsie. (Soc. de Neurol.) *Rev. Neurol.*, juillet 30, 1906, p. 702.

## GENERAL AND FUNCTIONAL DISEASES—

- Chorea.—HERBERT FRENCH. Chorea Gravidarum. *Practitioner*, Aug. 1906, p. 178.
- Epilepsy.—LEUBUSCHER. Epilepsie mit Halbseitenerscheinungen. *Neurol. Centralbl.*, Aug. 16, 1906, S. 738.
- EPILEPSY AND CRIME. Leading Article. *Brit. Med. Journ.*, Aug. 4, 1906, p. 284.
- POLLAK. Ueber Lumbalpunktion bei Eklampsie. *Zentralbl. f. Gynäkol.*, Aug. 4, 1906, S. 865.
- Neurasthenia.—JENDRASSIK. Über Neurasthenie. Breitkopf & Härtel, Leipzig, 1906, M. —75.
- JENDRASSIK. Über Neurasthenie. *Sammlung klin. Vorträge*, Juli 1906, S. 657.
- POECHE. Geschlechtliche Neurasthenie und andere sexuell-nervöse Schwäche und Erschöpfungszustände. Fiedler, Leipzig, 1906, M. 2.40.
- BATUAUD. La Neurasthénie génitale féminine. Maloine, Paris, 1906, 4 fr.
- Myasthenia.—BEDEDETTI. Astasia-abasia traumatica in bambina epilettica. Manicomio, Anno xxii, No. 1, 1906, p. 94.
- Tetany.—FRANKL-HOCHWART. Die Prognose der Tetanie der Erwachsenen. *Neurol. Centralbl.*, Aug. 1, 1906, S. 694.
- Torticollis.—LEROUX. Les torticolis d'origine otique. *Presse méd.*, août 4, 1906, p. 495.
- Sleeping Sickness.—GRANT. Sleeping Sickness. *Montreal Med. Journ.*, July 1906, p. 452.
- Exophthalmic Goitre.—FISCHER. Herneurosen und Basedow. *Münch. med. Wchnschr.*, Aug. 7, 1906, S. 1568.
- SKLODOWSKI. Beitrag zur Behandlung Basedow'scher Krankheit mit Röntgenstrahlen. *Deutsche med. Wchnschr.*, Aug. 16, 1906, S. 1340.
- JACOB. Pathologie und Therapie des Morbus Basedowii. *Ther. Monatshefte*, Juli 1906, S. 317.
- SCHULTZE. Zur Chirurgie des Morbus Basedow. *Mitt. a. d. Grenz. der Med. u. Chir.*, Bd. 16, H. 2, 1906, S. 161.

## MISCELLANEOUS SYMPTOMS—

- NÄCKE. Sind die Degenerationszeichen wirklich wertlos? *Vierteljahrs. gericht. Med.*, Juli 1906, S. 45.
- TSIMODA und SHIMAMURA. Beiträge zur pathologischen Anatomie der sogenannten "Katayama-Krankheit" zur Ätiologie der Hirngefäßembolie und der Jackson'schen Epilepsie. *Wien. med. Wchnschr.*, Aug. 18, 1906, S. 1682.
- I. OHM. Beitrag zur Klinik der Zwerchfell-lähmungen. *Ztschr. f. klin. Med.*, Bd. 59, H. 5 u. 6, 1906, S. 521.
- JOURDRAN. Du tremblement palustre. *Presse méd.*, août 15, 1906, p. 518.



- L. KAST. Zur theoretischen und praktischen Bedeutung Head'scher Zonen bei Erkrankung der Verdauungsorgane. *Berlin. klin. Wchnschr.*, Juli 30 u. Aug. 6, 1906, Sn. 1033, 1070.
- G. LENTHAL CHEATLE. The Relation between a Cutaneous Nævus and a Segmental Nerve Area. *Brit. Med. Journ.*, Aug. 18, 1906, p. 363.
- LEWANDOWSKY. Ueber Projektion der Schmerzempfindung von der unteren auf die obere Extremität bei Herd im Dorsalmark. *Centralbl. f. Nervenheilk. u. Psychiat.*, Aug. 1, 1906, S. 593.
- GÜLLMANN. Eine zweckmässige Methode, den Patellarreflex zu prüfen. *Fortschr. der Med.*, Juli 20, 1906, S. 615.
- NOICA. Etude sur l'antagonisme des Réflexes Cutanés et Tendineux dans les Paraplégies Spasmodiques. (Soc. de Neurol.) *Rev. Neurol.*, juillet 30, 1906, p. 703.
- NICOLA CASILLO. Il fenomeno di Babinski. *Ann. di Neurologia*, Anno xiv., f. 2-3, 1906, p. 181.
- IOTEYKO. A propos de récents travaux sur l'auscultation du muscle dans les paralysies, la contracture et la réaction dégénérescence. Quelques considérations sur la théorie motrice du sarcoplasme. *Journ. de Neurol.*, juillet 20, 1906, p. 272.
- MARCHENAND. Double pied bot paralytique; varus à droite, valgus à gauche. *Arch. de Méd. Navale*, août 1906, p. 102.
- PONCET et LERICHE. Pathogénie des ankyloses spontanées et particulièrement des ankyloses vertébrales. *Presse méd.*, août 4, 1906, p. 482.
- MAUDSLEY. Brachioptegia of Cerebellar Type and Rhythmic Tremor, with an Attempt to Explain the Symptoms and to Localise the Lesion. *Intercolonial Med. Journ. of Australasia*, June 1906, p. 302.
- BABINSKI. Asynergie et Inertie Cérébelleuses. (Soc. de Neurol.) *Rev. Neurol.*, juillet 30, 1906, p. 685.
- RODOLFO BONFIGLI. Contributo clinico ed anatomo-patologico allo studio dell' afasia. *Riv. di Patol. nerv. e ment.*, Vol. xi., f. 6, 1906, p. 266.
- BOULENGER. Quelques considérations sur l'écriture en miroir. Les troubles de l'orientation et son éducation. (Suite.) *Journ. de Neurol.*, juillet 20, 1906, p. 261.

#### TREATMENT—

- STEYERTHAL. Die Ernährung Nervenkranker. *Ztschr. f. Krankenpflege*, Juli 1906, S. 241.
- BRISAUD, SICARD, TANON. Essais de traitement de certains cas de contractures spasmes et tremblements des membres par l'alcoolisation locale des troncs nerveux. *Rev. Neurol.*, juillet 30, 1906, p. 633.
- LÖHRER. Zur Behandlung hysterischer Kontrakturen der unteren Extremitäten durch Lumbalanästhesie. *Münch. med. Wchnschr.*, Aug. 7, 1906, S. 1568.
- KIRCHNER. Apparat zu Operationsübungen am Schläfenbeine. *Ztschr. f. Ohrenheilk.*, Bd. 52, H. 1-2, 1906, S. 90.
- ADAM. Des établissements d'aliénés, d'idiots et d'épileptiques. Du rôle du médecin dans ces établissements. *Ann. méd.-psychol.*, juillet-août 1906, p. 45.
- DEL GRECO. Sul trattamento morale dei delinquenti pazzi. *Manicomio*, Anno xxii., No. 1, 1906, p. 90.

# Review of Neurology and Psychiatry

---

## Original Article

### TYPES OF THE DEVOLUTIONAL PSYCHOSES.<sup>1</sup>

By CLARENCE B. FARRAR,

Assistant Physician and Director of the Laboratory, Sheppard and Enoch Pratt Hospital ; Assistant in Psychiatry, Johns Hopkins University.

#### I.

As a foreword to the subject of the Epochal Psychoses, one phase of which claims our attention in the present discussion, I should like to refer to several points of view from which the attempt has been made to interpret forms of insanity in general.

We have heard much of the warfare of the so-called *symptomatologic* and the *clinical* methods, and their varying fortunes are familiar to all. Of necessity the symptomatologic method came first, and until the emancipation of psychiatry from the blighting control of theology and the uncertain authority of speculative philosophy was guaranteed, it represented the only avenue of approach. In its most primitive form the symptomatologic method recognised but very few distinct diseases of the mind ; these were assumed to be determined by the most conspicuous symptom in the disease picture, and naming this symptom constituted the diagnosis. Etiology, course, and outcome of the psychosis were of secondary importance in so far as the identification of the pathologic condition of the hour was concerned. To what errors the symptomatologic method may

<sup>1</sup> Presented at the Toronto Meeting of the British Medical Association, 23rd August 1906.

lead is shown in the attempt of LASÉGUE to construct out of his *délire de persécution* a disease entity from the symptom picture alone without reference to subsequent course or termination. Indeed LASÉGUE expressly stated that he had purposely avoided taking account of the fate of his patients, declaring that the disease was characterised and determined by the symptom complex of a certain stage. It is this view, often unconsciously followed, which has stood in the way of accurate discrimination and the deeper appreciation of the nature of mental disease processes even up to the present day.

The beginnings of psychiatric differentiation we nevertheless owe to the symptomatologic method. It furnished the stepping-stone for the work of PINEL and ESQUIROL in launching the modern science, and culminated finally in the masterpieces of symptomatologic psychologic analysis of WERNICKE the pilot-alienist, who died last year.

The clinical method was the outgrowth of the nineteenth century. It had been discovered that the peripheral appearances, for example of mental depression or exaltation or of psychic enfeeblement, such as had stood for the earlier symptomatologic entities, were not sufficient for the determination of individual diseases, and that indeed these several states might occur in the most varied conditions, differing widely from each other in etiology, course, and event. The clinical picture therefore began to take a biographic perspective of mental diseases. The method was new and the way beset with diagnostic difficulties, and in the face of these the extreme conclusion was reached that there was but one form of mental alienation, the four-stage insanity of ZELLER, of which the conditions of depression, excitement, and weakness represented only different symptomatologic phases.

From this confusion of despair of the possibility of clinical differential diagnosis there was, however, soon a recovery in the attempt of distinguished clinicians in various countries to separate from the chaos of material certain so-called typical disease forms, taking into account the personal and family antecedents, the various etiologic factors, the entire course of the disease, and the fate of the patients.

The clinical method has resulted thus in the setting up of a number of pathologic types, such as the *folie circulaire* of FALRET, or the *folie à double forme* of BAILLARGER, the German

successor of which is the *manisch-depressives Irresein* of KRAEPELIN; the *Vesania Typica* or *Katatonie* of KAHLBAUM, and the *Hebephrenie* of HECKER, the former being the natural successor of the four-stage insanity of ZELLER; the *Amentia* of MEYNERT; the *démence précoce* of MOREL and CHRISTIAN, and the *Dementia Präcox* of KRAEPELIN.

Assuming as it does that the course and outcome are the determining factors of the psychosis in contradistinction to the symptomatologic method which looks primarily at the disease state as it exists, the clinical method has been aptly described as offering a *longitudinal section* of disease, while the symptomatologic method offers only a *cross section*.

The growth of the clinical method was the best thing in psychiatry which the nineteenth century had to offer, and it brought with it sanguine expectations not destined to be entirely realised.

With the differentiation of certain typical disease patterns which large numbers of patients were found to fit by observers widely distributed, the hope was not unnatural that as knowledge accumulated and differentiation became still more accurate, new and independent forms would gradually be discovered, as a result of which the great bulk of undiagnosed material would finally be used up, each case being assigned in the course of observation to its proper pigeon-hole, each with its label attached.

There are many who have realised that this psychiatric millennium is an impossibility, and who, seeing the decline and fall of previously accepted conceptions of disease entities and their replacement by others which tend to undergo the same fate, have become more or less discouraged, just as did the observers alluded to of the mid-nineteenth century, of ever arriving at even an approximate classification of mental diseases.

It has been urged against the writers of text-books of psychiatry that they concentrate their attention upon the so-called typical cases, leaving the obscure cases to their obscurity. Of these the attempt has been made to dispose of many by describing them as mixed forms, intermediate or transitional forms, allied forms, combined forms, etc., *ad infinitum* of the assumed typical forms. By these subterfuges the clinical method in the strict sense expresses its disappoint-

ment, and the question of purity and adulteration of psychoses becomes a serious one. Thus the position of the classification sceptics is strengthened.

But to classify is not in itself the aim and end of psychiatry. The excursion of the pendulum has covered fifty years, and there are signs that the point of the return swing is near. Already clinicians are taking a broader view of mental pathology than that which attempts to discover in every patient some definite disease-form.

This broader view may be described as the *biologic method*. It comprehends the advances that have been made by both the symptomatologic and clinical methods, but its standpoint is less dogmatic. It looks at diseases of the mind not so much as stationary or transitory symptom-complexes, nor indeed alone as protracted manifestations of individual pathologic processes, it looks rather at the diseased personality in its entirety. It includes therefore both the symptomatologic and the clinical pictures and something more. It is neither a cross section nor a longitudinal section of disease, but embraces both, and takes the cubic measurements into the bargain.

The biologic method studies personality first and disease second; and not despairing at the ultimate futility of absolute clinical differentiation, it turns rather to the minute analysis of the perverted functions of individual minds, comparing them with each other point by point, both in health and disease. Under the influence of this conception, whatever further growth the symptomatologic and clinical methods are capable of will proceed to the best advantage.

## II.

Turning now to the Epochal Psychoses, we find that they are a relatively modern product. The symptomatologic classification of cases of depression as *melancholia*, and of cases of excitement as *mania*, drew no fine distinctions between such conditions appearing in youth, in adult life, or in old age, and epochal differentiation did not go beyond the recognition of the terminal mental reduction of the aged.

But with the clinical observation that the course and outcome of psychoses in many points symptomatically similar might

differ with the time of life, and that the critical periods favoured the development of particular forms of alienation, there arose the concepts of the so-called *adolescent* and *climacteric insanity*. In the latter the epochal physiologic changes were assumed to stand in direct etiologic relationship with the mental derangement, and many varied forms were classed together under the general head of climacteric insanity which was found not to be limited to the female sex, but to occur in men as well, at a corresponding or somewhat more advanced age.

The pathologic mental conditions of the grand climacteric are not, however, those of the senium proper, and we are thus accustomed to separate the psychoses of later life into two groups—the *senile* and the *präsenile* forms, although the distinction does not always hold. That the age of the patients is not the distinguishing factor is shown by the fact that one may show evidences of senile decay at forty-five or fifty, while a second passes through an affect psychosis with recovery at seventy. ESQUIROL described two cases in the Salpêtrière of maniacal attacks in women over eighty years old who got well.

The earliest of the devolutional psychoses appear as a rule about the forty-fifth year, and fixing upon this as a purely arbitrary boundary we shall have a period of a decade and a half or two decades during which occur morbid states predominatingly of a depressive type, many of which recover, and beyond which appear by preference the conditions with distinctly senile colouring.

It is obvious that all the psychoses occurring in the devolutional period are not necessarily of the devolutional type, and we must distinguish the *accidental* cases from the *epochal*. Under the former heading are to be included those cases which are known to occur at widely separated ages. For example, well characterised attacks of maniacal-depressive insanity, with or without previous phases of illness, may occur within this period. Isolated cases of late katatonia are also not unknown. Various intoxication and infection psychoses, the traumatic neuroses, the different organic and exhaustion states, amentia, hysteric and epileptic insanity—all of these may occur during the devolutional period as well as at other times of life.

Of the organic diseases, *paresis*, which appears in the majority of cases after a certain length of time following specific infection,

occurs for the most part during the earlier prime of life. It may, however, for patent reasons also develop during the devolutional period, and in such instances is to be looked upon as an accidental psychosis, not dependent upon the regressive changes peculiar to the epoch. With the *cardio-vascular psychoses* the question is different. Arterio-sclerosis is in the bulk of cases the accompaniment of later life, and the pathologic mental states dependent upon it are therefore in a sense specific for the advanced devolutional period.

The insanity of arterio-sclerosis appears under a variety of forms which seem to be the direct expression of the structural and nutritional changes in the central organs. On the other hand nothing is more common in the senile and *präsenile* types than that arterio-sclerosis should be present as a complicating factor without being the sole etiologic agent, and it is sometimes possible in such cases to trace the influence of the cardio-vascular condition in modifying the manifestation of the psychosis. But whether as primary disease or symptom, the separation of the morbid mental states of arterio-sclerosis constitutes one of the most important differentiations among the devolutional psychoses.

A second group of cases comprises the *involutional sexual phrenopathies*, including perhaps the *präsenile persecutory forms* (*präsenile Becinträchtigungswahn*, KRAEPELIN).

A third form is represented by the *presbyophrenia* of WERNICKE.

With the mention of these types we shall pass at once to the group in which our present interest centres—the *devolutional depressive forms*.

First comes the condition of affect-depression known as *melancholia*. Various observers have noted that the depressive psychoses in old people run a somewhat different course and depart symptomatologically from the depressive states of younger individuals, and have thus distinguished between the melancholia of later life and that of earlier years; but this viewpoint has become most conspicuous through KRAEPELIN, who draws a hard and fast line between the two conditions, and reserves the term melancholia exclusively for the involutional cases.

The melancholia of KRAEPELIN is still a distinctly wide-mouthed receptacle in spite of the fact that the disease-concept is a very much narrower one than that of the melancholia of the

symptomatologic school. KRAEPELIN includes under melancholia "all pathologic conditions of anxious depression in later life, which do not represent phases of other forms of insanity." The breadth of the category is adequately expressed in the definition. It comprises cases of quiet affect depression (*melancholia simplex*); active cases with marked apprehensive anxiety (*melancholia activa vel agitata*), which may culminate in *raptus melancholicus*; and finally, fantastic depressive delusional conditions, and even continued forms accompanied by depression and ending in progressive senile deterioration. It is in this broad field of symptomatologically heterogeneous conditions that we are endeavouring to discover types which may possibly be clinically distinct.

As to the relations between melancholia and the depressive phases of maniac-depressive insanity, two conditions which KRAEPELIN wishes to see clearly distinguished as independent and individual diseases, there exist unreconcilable differences of opinion, and there are not a few who are unable to see in involutional melancholia anything else than one of the manifestations of maniac-depressive insanity, modified, perhaps, by the time of life. The peculiar ideas of sin, unworthiness, and future punishment which constitute one of the important symptoms of the involutional cases, are by no means always present, and may also be encountered in young individuals. The same is true of the anxious affect state. Moreover, one of the emphasised diagnostic criteria of maniac-depressive insanity or circular depression, that of periodic recurrence, is not at all uncommon in involutional depression.

One would doubtless be near the truth in saying that involutional melancholia represents a condition of psycho-motor and affect depression, such as may occur at any time of life, and that its peculiar colouring, by which it unquestionably differs from depressive cases in earlier life, is due simply to the epoch. While therefore in these cases we confidently look for certain symptoms which are much less likely to be in evidence in younger individuals, and which may be said therefore to be in a certain sense specific, they are specific nevertheless only in the sense in which the old man by reason of natural structural and functional alterations in the central organs acts and thinks differently from the young adult. Thus, while during the earlier



and mid years of life the affect psychosis of choice is maniac-depressive insanity, and during the later years a more or less specific form of depression occurs, yet some of the symptoms of this latter disease are not absent from youthful cases, and correspondingly, definite attacks of maniac-depressive insanity are not infrequent in the devolutional period.

The assumed clinical distinctions are rather biologic differences, and are closely associated with the psychologic metamorphosis of age. This view is further supported by the facts that in one instance a well-defined attack of circular depression may appear for the first time in later life and present the pathognomonic features of the psychosis—subjective insufficiency with affect depression, and psycho-motor inhibition,—while in a second case of maniac-depressive insanity with recurring depressive attacks, the later phases may show unmistakable epochal modifications and take on more or less the complexion of involutional melancholia.

It is even possible to trace the metamorphosis. Take the feel of *subjective insufficiency* of circular depression. The normal adult lives in tense relation with the objects of his environment, he maintains himself in a state of ready adaptability, reactions follow quickly and easily, but nevertheless as a result of deliberation or at least with conscious approval. If now the ready succession of the psychic elements be functionally interfered with and consciousness remain unclouded, a subjective feel of insufficiency is the inevitable result.

In the patient overtaken in the devolutional period on the other hand, with his waning relational tenseness and weakening powers of adaptation, whose reactions are rather those of habit and custom than of conscious deliberation and logical choice, under the influence of pathologic depression the subjective insufficiency becomes subconscious and is reflected in a conscious uncertainty and insecurity, which are the essential conditions of the anxious apprehensive states of certain of the devolutional cases.

Further, the objective symptom of *psycho-motor inhibition* in the circular cases is significant not only of a psychosis but of a time of life as well. Psycho-motor inhibition is the outward expression of the resistance of an habitually active organ to hindering influences of a functional nature, it is the counterpoint

of the life of action which struggles to make good, it is analogous to the brake on the wheels of an engine at full steam. It is therefore in a sense a positive symptom.

In the old man on the contrary the gauge is low, and the wheels tend to slacken their speed, so that in the presence of pathologic depression the effect of the brake is less conspicuous, although not entirely absent, and there may thus result a condition of apparent indifference or even apathy which is characteristic of a group of devolutional cases. The positive symptom has been replaced by a negative one.

These observations, while in no wise under-reckoning the value of the clinical differentiation of epochal depressions, emphasise nevertheless the biologic consanguinity of these affect states at different times of life.

### III.

In the great group of depressive psychoses of the devolutional period there are unquestionably various sub-types, if not distinct clinical forms, and with a view to ascertaining in how far such a differentiation is justifiable, we have been following carefully in the Sheppard-Pratt Hospital in Baltimore a number of the patients belonging to this group.

Our results, which thus far are imperfect, only partial, and thoroughly provisional, I have summed up very briefly and somewhat too schematically in the accompanying chart (p. 683). An absolutely complete representation of the facts in such a chart would defeat its purpose in making it too wordy and complicated. In the present instance we have merely seized upon the important points as they appeared to us, and emphasised them. It must of course be understood that these observations are in an entirely fluent state, and have nothing to do strictly with what might be spoken of as final conclusions.

A trite remark may nevertheless here be accented, namely, that in the symptomatologic analysis of cases there is no single characteristic which is pathognomonic of or limited strictly to any one disease-type. In the conditions we have been studying certain symptoms run through them all, although in varying degrees of intensity, some features being exaggerated in one condition and inconspicuous in another, while other symptoms on the contrary are so constant and so pronounced in a given

disease-state, that taken together they lend a certain provisional definiteness of outline and colour to the picture.

Our cases have been analysed with regard to about one hundred and fifty of the more important normal and abnormal elements of the mental life and physical status of the devolutional period. In our chart only a very few of the particularly representative elements have been indicated, and, as will be seen, they concern exclusively the psychic sphere. Other points will be referred to in discussing the individual conditions.

#### IV.

Under *true melancholia* we have included a fairly definite and well circumscribed clinical picture, with confines distinctly narrower than those of the involutorial melancholia of KRAEPELIN. It comprises cases which previous to the forty-fifth year are reported to have been mentally sound, but which during the fifth or sixth decade pass through one, sometimes two attacks of affect depression lasting one or more years. Neither sex is spared. In women the psychosis may be associated with or soon follow the menopause. In such cases the patient may get well and a number of years later suffer a recurrence from which she may or may not recover. The depression is unaccompanied by maniacal phases or symptoms.

Melancholia as here understood is characterised by fairly normal sensation and orientation, an affect depression which may lead to *odium vitæ* and suicidal acts, a narrowed egocentric association, thought processes tolerably active within their confined circle, introspection, self-abasement, auto-accusation, exaggerated ideas of sins committed, often the unpardonable sin, and of future damnation. The chief lesion of consciousness is in the idea of the Ego, and the disease may therefore be designated as an *Autopsychosis*. Associated with this primary lesion, somatopsychic delusions, expressed as ideas of change and derangement in the patient's physical economy, are not uncommon. Consciousness is blackened by a deep-dyed religiosity; the patient is too wicked and vile to be tolerated among men, to be given food and drink, or even to be seen. He has sinned basely against his family, against mankind, and against the Most High. He has violated all the Commandments, his vileness reeks to heaven, his body will be destroyed by the most exquisite torture,

and his perjured soul committed to the everlasting flames. Indeed, to make a realistic comparison, the whole picture is quite such as a susceptible individual might be expected to have presented after listening to the sermon of JONATHAN EDWARDS on "Sinners in the Hands of an Angry God."

In all of these beliefs the patient displays a striking *subjective certainty* which admits neither of doubt nor of argument. It is the time of life to be dogmatic, and this tendency is reflected actively in the psychosis.

The depth of the lesion is shown in the *autognosis*. Insight is very defective. The patient is persuaded that he is in full mental health—it is his soul which is lost, not his mind. He would to God it were nothing worse than a disease of the mind, but conscience convicts him otherwise.

In this condition the feel of subjective insufficiency and incapacity common in the circular forms is likely to be inconspicuous or absent. The patient may believe himself quite capable of resuming and carrying on his usual occupation, and his reasons for not doing so are bound up in his autopsychic delusions.

The *psychic inhibition*, which is at the bottom of the sense of circular subjective insufficiency, is not a prominent symptom in melancholia vera during the acme of the psychosis, while in the circular cases it is exactly at the height of the disease that it is most pronounced. During the prodromal and early stages of melancholia a mild degree of inhibition is common, and this is the rule in fact, with all cases of affect depression; but with the development of the psychosis and the unfolding of the autopsychic ideas with concomitant egocentric narrowing of consciousness, the pathologic thought processes tend to become more and more fixed and habitual, and flow in their limited channels with the readiness of settled convictions, in the presence of which psychic initiative and deliberative choice are conceived not only as futile but unnecessary.

Finally, it is to be noted that in the cases under discussion a condition of the effect expressed as *anxiety* or apprehensiveness is seldom entirely lacking. It may appear only occasionally and in a mild form, or may reach such a degree of intensity as to make life very distressing to the patient. This anxiety is always recognised and understood by the subject as such, and is

the logical outgrowth of the depressive content of consciousness with its stamp of absolute subjective certainty. It may be considered therefore in most cases rather as a secondary symptom psychologically determined than as a primary manifestation pathologic *per se*. The whole situation is perfectly illustrated in a remark which the physician so frequently hears from patients of this class—"you too would be worried and afraid and anxious if you had such thoughts as mine *and knew that they were true*." The anxious affect does not depend upon clouding, disorientation, or hallucinosis. It is the natural expression of the dark thoughts which dominate consciousness, and is strongly coloured with pathologic remorse.

## V.

If now we look at the second group of cases, representing a condition which for convenience' sake may be spoken of as *Anxietas Præsenilis*, we find a situation essentially different from that in melancholia vera. The disease occurs a little later in life, usually in the sixth or seventh decade, and the great majority of the patients are women. The morbid process is deeper-seated and severer, and the outlook decidedly more dubious. It represents more specifically the regressive changes, both structural and functional, of later life.

Both primary and secondary sensation are involved, these terms being used in the sense of the primary and secondary identification of WERNICKE, and a degree of disorientation is therefore the rule. Fallacious sensation is often present, usually in the form of sensory misinterpretations, but fleeting isolated hallucinations of hearing and sight may also occur.

The psychosis is pre-eminently the expression of severe defect in the power of mental assimilation and adaptation, and undoubtedly rests upon a considerable diffuse cortical change. Arterio-sclerosis is a not infrequent complication.

With the diminished power of quickly and correctly interpreting sensory impressions and reacting to them, there is born a condition of *subjective uncertainty* and insecurity, a sense of strangeness, not-at-homeness of the Ego in the presence of its environment. We see here a lesion in the relational sphere of consciousness, or the *allopsyche*, and in this sense the

condition may be spoken of as an *allopsychosis*. To the patient the surrounding objects of the material world acquire a character of unnaturalness or even unreality, and this sense of objective strangeness and unreality may extend to include the patient's own body. Thus the Ego finds itself lost and adrift in a sea of sensory unrealities and misinterpretations. These are uniformly of a depressive character, determined primarily no doubt by the dysphoria of organic ill-functioning of the devolutional period. From the subjective uncertainty and objective unreality there come fear and vague alarm, an involuntary and uncontrolled sense of apprehension in the face of every new impression. In proportion as the affect sphere of consciousness is active, this cloudy apprehensiveness may develop into a veritable panic of agonised fear with lively motor agitation and a tendency to self-mutilation. This *status anxietatis* may persist with slight remissions for weeks or months, and represents the most essential character of the psychosis.

The condition is utterly dissimilar to the anxiety referred to in connection with melancholia. There the feeling expressed a dread of awful things to come, clearly conceived and subjectively certain, complicated with anxious remorse for awful things in the past of which the patient accused himself of being the author. The situation was therefore one of apprehensive fear of the awful *certainities* of existence. Here, on the contrary, the feeling is one of dread and anxiety less of the future than of the present, alarm before the unknown, uncomplicated with remorse; it is a fear of the awful *uncertainty* of existence.

A further important distinction between true melancholia and the condition here described lies in the character of the content of consciousness itself. In *anxietas præsensilis* autopsychic ideas such as were described in melancholia play no part. There is no auto-accusation, no self-abasement, no alloistic self-blame. Religious delusions with remorse and dread of future punishment do not enter the scheme. Indeed, in spite of their uncertainty, patients of this class regularly reply in answer to questions that they have no particular regrets regarding the past, and that they have done nothing for which they should suffer.

The autognoses of the two conditions placed over against each other well illustrate their differences.

From the melancholiac we should perhaps get something like this:—"I have no complaint to make, I know my condition, my mind is clear. My body is diseased from my evil life and my soul is in torment, but I deserve it all, and what I now suffer isn't a circumstance to what is still in store for me. I have been false, unnatural and wicked from my birth, the most sinful creature alive, and am to be made an example before the world. It is true. God has revealed it to me through the voice of conscience."

From the *anxieuse* we should have this:—"I don't know what's wrong with me; I can't get right; nothing seems the same to me any more, and I always have such fears, such awful anguish. I don't know what makes it. I never did any one any harm. I don't know where I am. I don't know what to do, but this is not the right place: I don't belong here. Don't let any one kill me."

The facial expression of the *anxieuse* reflects the two chief elements—psychic uncertainty and fearful apprehension, displayed in the characteristic frontal *omega contraction*.

With the development of the psychosis the mental horizon may become progressively narrower. Initiative in thought and action is *nil*; all psychomotor activity assumes more and more the quality of the habitual; the habitual becomes the reflex; the reflex may become automatic. The laws of inertia hold, and reflex movements once begun tend to continue. Thus we get the symptoms of *verbigeration* and oft-repeated, stereotyped motions of the body or its members, and these finally may assume a definite *rhythmical form*. These movements are thoroughly characteristic. They arise sometimes as a result of stimuli from without, at others with no discoverable cause, and still again from demonstrable processes going on within the patient's body. Thus, for example, during a state of severe anguish the respiratory rate is accelerated, and at the same time waving or beating movements of hands or arms may appear, keeping time with respiration; or the patient may utter an ejaculation of despair to the same rhythm, pronouncing the same word or phrase with identical intonation time after time with each respiratory excursion.

The subjective uncertainty and the tendency to reflex rhythmical expression favour the development of a degree of

*suggestibility* such as would be quite foreign to the true melancholiac. Thus in certain cases the automatism of verbigeration may be interrupted by the observer, and the set phrases of fear replaced by others at suggestion, even quite contradictory to the former expressions and out of keeping with the affect tone. This phenomenon, in which the general bearing and the countenance reflect uncertainty and anguish, while on the lips is a phonographically repeated phrase of confidence and joy, is very striking indeed.

One or two concrete examples of the character of the rhythmical verbigeration in *anxietas præsens* may still be given. They display at the same time perfectly the fundamental characteristics of the psychosis. One woman in whom the sense of subjective uncertainty was extremely pronounced, repeated frequently this phrase over a period of four years,—

“I don't know where to go, nor what to do ;  
Nor what to do, nor where to go,”

giving to the words a chanting rhythm which never varied.

Another female patient, a German, chanted in the same manner the following:—

“Ich weiss nicht wo ich bin,—  
Und wer ich bin.—  
Und was ich bin,—  
Und wie mir ist,”

During the earlier part of the course of the disease the anxious affect tone may be fairly constant, merely showing variations in intensity. Later there appears to be a tendency to remittance, or indeed an *alternation of states*, in which periods of acute anguish with sensory misinterpretation, motor unrest and rhythmical verbigeration, alternate with intervals of quiet in which the face is relaxed and the patient lies tolerably motionless, inaccessible, even mute.

Sleep is as a rule very much better with these patients than with melancholiacs, and the appetite may even be ravenous.

The tendon reflexes are regularly exaggerated, sometimes greatly so. The pupils are usually small and slightly sluggish. Temperature is a trifle subnormal, pulse moderately accelerated in most cases, and the blood pressure elevated. A varying degree of arterio-sclerosis is common. In some instances a definite



parallelism can be demonstrated between the intensity of the anxious effect and the height of the blood pressure, treatment directed toward lowering the blood pressure and pulse rate also relieving to a degree the pathologic affect. While therefore in many instances the connection between cardio-vascular condition and mental state is unmistakable, yet this does not hold for all, and in typical cases such as we have been describing, the arteriosclerosis should perhaps be considered only as a complicating factor.

It is impossible here to outline completely the clinical picture of *anxietas præsenilis*. There will be occasion to recur to the subject later, and we shall therefore leave it for the present with this preliminary sketch. It has seemed to us to be clearly distinguishable from melancholia proper, although most authors mention its characteristics, or part of them, only in common with those of the latter disease. The prognosis is distinctly less favourable than in melancholia vera.

## VI.

There remains to refer briefly to a third form of devolutional depression which differs in certain essential features from both of the previously described types. It appears to attack men by great preference, and in its general character may be described as an *apathetic psycho-motor depression*.

Individuals who all their lives have been accustomed to arduous physical toil, or sometimes close mental application with the worries of commercial life, and who continue their labours unmodified or perhaps with increasing responsibilities into their later years, sometimes sink gradually during the devolutional period into a state of inertia, indifference, and mild depression, withdrawing from their usual occupation and becoming taciturn, unsociable, morose, or even resistively perverse. Occasionally the onset appears to be fairly sudden after some unusual exertion, exposure, or worry, when the patient declares that it is of no use any longer, he cannot keep up the unequal struggle, he has played his game out, he is used up and done for.

We have to do here undoubtedly with the expression of accumulated *fatigue* through years of monotonous labour, with insufficient variation, recreation, and recuperation. The actual

situation is often fairly appreciated by the patient himself, and his autognosis may be tolerably accurate, although his mental state does not allow him to seek explanations or trace connections between causes and effects.

In the feeling of *subjective insufficiency* we see a symptom not encountered in either of the two forms already described, but common in the affect depression of circular insanity. To this latter type, however, the apathetic depression of senescence bears little resemblance. It presents no suggestion of maniacal symptoms, either as phases or complications; it shows no vivid affect, and the psycho-motor inhibition of the circular cases is replaced by a simple psychic depression. It is not so much a question of the overcoming and suppressing of a tendency to action such as would be characteristic of earlier years; it is rather a more or less complete lapsing of the action impulse altogether. The change is thus pre-eminently a biologic one, as has already been suggested; indeed the *ensemble* in *depressio apathetica* impresses one as negative, both subjectively and objectively.

Sensory falsifications play little or no part in the development of the psychosis. At the beginning the patient may entertain vague ideas of harm befalling him, but they are uncertain and fleeting, and do not lead to the development of an anxious state. He may even be mildly self-accusatory in that he upbraids himself for his sloth. For the most part, however, he remains quiet and uncommunicative, expressing neither hope, nor fear, nor desire, although commonly enough a mild degree of nostalgia is present even though not actively voiced.

It is the rule to observe in the condition we are considering a certain amount of *subjective uncertainty*. This, it will be remembered, is one of the cardinal symptoms of *anxietas præsensilis*. The setting of this element is nevertheless quite different in the two psychoses. In *anxietas præsensilis* the sense of uncertainty is closely associated with a degree of disorientation and mental anguish, usually with conspicuous disturbance of secondary sensation, and unaccompanied by well-marked conscious insufficiency. In *depressio apathetica*, on the other hand, sensation and orientation are usually intact, and the uncertainty is accompanied by apathy instead of anxiety. Indeed, the fundamental lesion here is in the relational warmth of the Ego to its environment, in the patient's *interest* in life and its activities and

pleasures ; as a result of which, together with a fairly preserved insight, suicidal tendencies may become manifest, though they usually lack the energy of execution. Moreover, the failing *interest* and weakened voluntary attention bring it about that mental impressions are faint and easily lost. Objects of the environment are not vividly reflected in consciousness, and it results that the external world becomes to the Ego to a certain degree cold and colourless, distant, vague, or unreal. This sense of distance or vagueness is, however, distinct from the objective unreality of *anxietas præsenilis*. There this symptom was determined by misinterpretations with disturbed affect,—a *para-condition* ; here it is an element of diminished psychic activity,—a *hypo-condition*.

From all of these factors it follows that a certain defect of memory is seldom entirely missed in patients of this class, showing itself chiefly as a disturbance of the *recording faculty*. Of this defective recollection of current happenings the patients are themselves conscious, and it contributes its part to the feeling of uncertainty before mentioned.

In general bearing the patients are for the most part listless and indifferent. Their voluntary movements are few, slow, weak, without purposeful direction. Their power of initiative and decision is almost *nil*. Sleep and appetite are usually fair. Muscle tone is diminished, and the tendon reflexes are likely to be weak. Blood pressure and pulse are as a rule low, unless there be a marked degree of arterio-sclerosis.

The prognosis appears to be relatively good.

## VII.

In the foregoing discussion of certain clinical types of the devolutional psychoses the subject is by no means exhausted. So far as our material has furnished evidence it has seemed to us that these types were worthy to be considered separately, but, as has been said, our results are incomplete and distinctly provisional. What may be the ultimate relations of these various forms to each other, and to the senile psychoses proper, as well as to other possible undifferentiated forms, remains matter for continued observation.

	MELANCHOLIA VERA.	ANXIETAS PRÆSENILIS.	DEPRESSIO APATHETICA.
CHARACTER . . .	AUTOPSYCHOSIS	ALLOPSYCHOSIS	HYPOPSYCHOSIS
PRIMARY SENSATION .		X	
SECONDARY SENSATION		X	
HALLUCINOSIS . . .		X	
PSYCHIC DEPRESSION .			X
MOTOR DEPRESSION .			X
AFFECT DEPRESSION .	X		
APATHY . . . . .			X
ANXIETAS . . . . .		X	
SUBJ. UNCERTAINTY .		X	
AUTOACCUSATION .	X		
RELIGIOSE DELUSIONS	X		
SOMATOPSYCHIC DELS.	X		
SUBJ. INSUFFICIENCY .			X
VERBIGATION . . .		X	
INSIGHT . . . . .	POOR	PARTIAL	FAIR
SEX . . . . .	BOTH	FEMALE	MALE
PROGNOSIS . . . .	FAIR	DOUBTFUL	FAIR

## Abstracts

### ANATOMY.

#### AN INVESTIGATION INTO THE STRUCTURE OF THE LUMBO- (365) SACRAL-COCCYGEAL CORD OF THE MACAQUE MONKEY.

Miss M. P. FITZGERALD, *Proc. Roy. Soc.*, Series B, Vol. lxxviii,  
No. B 523.

THE present paper deals only with the section area of the grey and white substance in the various segments of the lumbo-sacral-coccygeal cord. A further paper is promised on the arrangement of the cell groups at the different levels, and a comparison with the human spinal cord.

J. H. HARVEY PIRIE.

#### THE NEUROGLIA FRAMEWORK OF THE CEREBELLUM. E. E. (366) SOUTHARD, *Journal of Medical Research*, August 1905.

WITH a large variety of material and by means of Mallory's neuroglia stain the author has sought to modify the hitherto dominant view of the neuroglia framework of the cerebellum with

particular reference to the histogenesis of Bergmann's fibres. He presents the total findings in cases of syphilis, ischemia, trauma, and bacterial infection, and concludes that the tissues of the cerebellum react in a characteristic way to injury. The Purkinje cell layer is a point of least resistance along which cleavage may easily occur, especially in macerating brains; moreover, these cells, together with their dendrites, suffer the maximum injury in impairment of the blood-supply or from bacterial infection. Severer injuries destroy not only the Purkinje cells but also the nerve cells of the granular layer, the neuroglia, however, persisting and reacting in correspondence with the nerve-cell degeneration.

The neuroglia reaction is characteristic of the several layers composing the folia. In the medullary centre the gliosis is a homogeneous feltwork. In the cortex the regular stratification is fairly definitely preserved. The Purkinje line of cleavage and degeneration is replaced with a line of neuroglia cells which produce numerous fibrils forming three layers—(1) a layer of fine fibrils lying flatwise to the outer limits of the medullary core; (2) superimposed and also lying flatwise to the medulla, but at right angles to the first layer, is another layer of fine fibrils. These two layers of fibrils parallel the contour of the cortex. (3) The third layer of fibrils is composed of coarser radial fibrils running vertically with respect to the medullary centre and passing out to the pia. These latter radial fibrils (Bergmann's) are the first to develop and arise from cells lying in Purkinje level, and not from cells of the inner layers, as revealed by the Golgi method. An apparent exception to this is noted in reference to the neuroglia reaction in a case of syphilitic marginal sclerosis of the cerebellum. The author's interpretation of the origin of the radial fibres in the syphilitic lesion is somewhat confusing and complicates the otherwise clearly described picture of the architectonic of the cerebellar neuroglia.

CHARLES I. LAMBERT.

**SUPPLEMENTARY PROCEEDING OF THE PYRIDINE METHODS  
(367) FOR THE RAPID DIFFERENTIATION OF THE RETICULUM OF NERVE ELEMENTS.** (Procedimento supplementare dei metodi alla piridina per la rapida differenziazione del reticolo fibrillare negli elementi nervosi.) DONAGGIO. *Riv. Sper. di Fren.*, Vol. xxxii., Fasc. 1-2.

THIS supplementary proceeding, described by Donaggio, which may be applied to his 3, 4, and 5 methods, is of value, not only because it shortens the time necessary for differentiation, but also because it assists in the demonstration of various morbid changes met with in human pathological tissues.

Although the nucleus and nucleolus are not stained by Donaggio's methods when applied to normal tissues, it has been found that in pathological material the nucleolus becomes differentiated into two distinct parts, a central portion, which has a pale blue colour, and a peripheral portion, which takes a violet colour. Those peripheral masses correspond to the basophile constituents of the nucleolus described by Levi.

This characteristic should be of the greatest value in examining human pathological material.

The various stages of these three methods up to the end of the staining process remain as described by the author in an earlier work, but, now, instead of putting the sections into water for a few seconds and then into spirit to differentiate, they pass from the staining fluid rapidly through water and then into an aqueous solution of pink-salt (1), (one part of concentrated solution of pink-salt to nine parts of distilled water), for one to five minutes; this solution should be made fresh each day, because it deteriorates rapidly.

The sections should then be passed into distilled water, which is changed several times, and care must be taken to see that the side of the coverslip opposite to that carrying the sections is cleansed, in order to avoid any precipitate; they are then immersed in spirit, and become differentiated in a few minutes. The length of time required varies with different sections; from two to five minutes is generally sufficient.

The spirit should only be used for a few sections.

They are passed into absolute alcohol; then to xylol; and finally the coverslips are cleaned, and the sections are mounted in neutral balsam.

The preparations should be kept in the dark.

(1) The author uses a concentrated solution of pink-salt (ammoniated chloride of tin), which may be obtained from C. Erba of Milan. *Note.*—It will be opportune here to correct a mistake which appeared in the description of method 5 which was published in the *Review of Neurology and Psychiatry*, February 1905. At the end of the second paragraph of page 88 is the following sentence:—"Now wash in water, changing it a few times, for 24 hours, and proceed to embed in paraffin." This should read:—"Now wash in water, changing it a few times, for two to four minutes, and proceed to embed in paraffin."

R. G. Rows.

**PSYCHOLOGY.**

**ON INSTINCT: A Psycho-Physical Study in Evolution and Dissolution.** (368) W. H. B. STODDART, *Journ. of Ment. Sc.*, July 1906, p. 491.

THE author maintains (1) that volitional and instinctive movements are performed by different motor tracts of the nervous system, the former by the cortico-rubro spinal representative of the pristine nervous system as it exists in birds, the latter by the pyramidal system; (2) that the volitional motor system, being evolved and developed later than the instinctive, is earlier and more readily affected in mental disorder; and (3) that when the instinctive motor system is attacked the instincts disappear in the reverse order of their development, and therefore of their evolution.

The development of the various instincts in the child is traced in some detail, and an instructive parallel is drawn between the stages of dissolution as seen in a general paralytic and the instinctive stages occurring in childhood.

In conclusion the author shows the importance of distinguishing between volitional and instinctive acts in medico-legal cases.

W. B. DRUMMOND.

**PATHOLOGY.**

**RETROGRADE DEGENERATION IN THE SPINAL NERVE** (369) S. WALTER RANSON, *Journ. Comp. Neurol. and Psychol.*, Vol. xvi, No. 4, July 1906, p. 265.

AN experimental investigation on the white rat. After section of the second cervical nerve it was found that one-half of the cells in the corresponding spinal ganglion disappeared. This ratio was found very constant in nine cases. Enumeration showed that many more cells had disappeared than could be accounted for in terms of medullated fibres cut at the operation. The number of fibres in the dorsal root is liable to greater individual variation, but on an average there was a loss of about 17 per cent, and the dorsal roots seemed more susceptible to degenerative changes in the young than in adult animals. The degeneration in the dorsal roots apparently cannot without some qualification be attributed to the degeneration in the spinal ganglia. The degeneration of fibres and cells was found not to be progressive but to be completed before the end of the first two months. A good bibliography is given along with a short summary of the literature.

J. H. HARVEY PIRIE.

**LESIONS OF THE NEURO-FIBRILLARY RETICULUM OF  
(370) THE NERVE CELL IN EXPERIMENTAL INANITION.**

(Lesioni del reticolo neurofibrillare della cellula nervosa nell' inanizione sperimentale.) RIVA, *Riv. Sper. di Fren.*, Vol. xxxii., Fasc. 1-2.

THE author has applied the methods of Donaggio to the nervous tissues of adult dogs in order to study the changes in the neuro-fibrillary reticulum which are produced by inanition.

The first dog was kept alive for 48 days without food during the winter months. On examination it was found that the reticulum in the cells of the anterior cornua were profoundly altered. The fibrils were disturbed and were arranged in vortices and spirals, and in irregular condensations. Some vacuolisation was also observed. In other cells, and more rarely, there was a rarefaction of the network in the centre of the cell, and various nodosities were present at different points of the reticulum; the long fibrils formed a marked condensation at the periphery of the cell.

The disturbance was most marked in the anterior cornua, less in the other cell groups. Vacuolisation was common, and in many instances the vacuoles were filled with a granular substance.

In the medulla and pons the same type of change was found, especially in the cells of the motor nuclei. Very little alteration was present in the cells of the cerebral cortex, and those of the posterior root ganglia were practically unaffected.

In the other animals experimented on these profound changes of the reticulum were absent, but many of the cells showed a marked degree of vacuolisation, even in some of the cell processes. This vacuolisation did not lead to much change in the surrounding reticulum.

The results obtained in these experiments suggest that the reticulum of the nerve cells suffers little injury from inanition when acting alone, and the more severe lesions, noticed in the first dog described, depended on the fact that the experiment was carried out in the winter, and the alterations were therefore the result of the combined action of cold and inanition.

R. G. ROWS.

**THE EFFECTS OF THE COMBINED ACTION OF FASTING  
(371) AND COLD ON THE NERVE CENTRES OF ADULT  
MAMMALS.** (Effetti dell' azione combinata del digiuno e del freddo sui centri nervosi di mammiferi adulti.) DONAGGIO, *Riv. Sper. di Fren.*, Vol. xxxii., Fasc. 1-2.

EARLIER researches of Donaggio and Fragnito have shown that the neuro-fibrillary reticulum of the nerve cells of the adult mammal



possess considerable powers of resistance to injurious agents. In the present inquiry the author deals with the changes produced in the reticulum of the nerve cells by the combined action of fasting and cold. Neither of the factors, when acting alone, produces any marked alteration in the fibrils of the reticulum, but, in animals which have been subjected to their combined action, marked lesions are constantly found.

In the rabbits examined by the author the cells of the grey matter of the spinal cord showed much change. In the cells of the anterior cornua the network was thinner than normal and intersected by large bands, which were uniformly and strongly coloured, so that there was no trace of any structure. Sometimes these bands were scattered throughout the cytoplasm; in other instances they were localised in the thicker reticulum around the nucleus; in other cells the meshes were much larger than normal.

The cells of the remaining grey matter showed even more serious alterations. The reticular structure had disappeared, and was replaced by large bands, often fusiform, running through the cytoplasm, and sometimes even into the cell processes. The distribution of these bands was quite irregular.

Similar changes of the endocellular reticulum appeared to a greater or less extent throughout the whole of the central nervous system, but the cells of the cerebrum were generally only slightly affected.

Vacuolisation of the cells, especially of those in the spinal cord, was frequently present.

Another interesting condition noticed was that, although the nucleus remained uncoloured, the nucleolus assumed a pale blue colour in its centre, while at its periphery lay three or four masses, which sometimes showed a granular structure, but much more frequently were stained uniformly violet. They correspond to the peripheral basophile contents of the nucleolus described by Levi. In the nerve cells of the normal animal these masses are not coloured by the methods employed in this research—the methods of Donaggio for staining the endocellular reticulum—and their presence is an indication of some pathological change which allows them to assume the violet colour. In one rabbit vacuolisation of the nerve cells was very frequently seen, and it was observed that they contained some substance in the form of short rods or sometimes in irregular masses which were similar to those described by Cajal in the vacuoles found in the nerve cells of animals which had died of rabies.

It has also been found that various toxines, which by themselves have little effect, can produce serious lesions of the neuro-fibrillary reticulum when combined with cold.

These researches also suggest that a greater importance should

be attached to the so-called rheumatic causes in the production of nervous diseases, and further, that the rheumatic cause is able to exert its influence only when it finds a toxic or infective-toxic basis on which to act.

R. G. ROWS.

**CONTRIBUTION TO THE PATHOLOGICAL ANATOMY OF  
(372) PARKINSON'S DISEASE.** (Contributo allo studio dell'  
anatomia patologica della malattia di Parkinson.) *Riv. di  
Patolog. nervosa e mentale*, Vol. xi., F. 4, pp. 145-70. Catòla.

THE author gives a schematic review of the remote and recent literature, and appends an extensive bibliography. There is little agreement as to what constitutes the specific anatomical basis of this disease. In general the findings have been those of cerebro-spinal senility, frequently of an earlier and severer character than is usual.

The author discusses at length the views held by different students—a few regarding it as a nervous state without an organic basis, others esteem it not as a clinical entity but a syndrome. He concludes with a critical *résumé* of the more recent neuritic and muscular theories. In the former there is lack of uniformity in the anatomical findings; in the latter, so far as observed, there seems to be considerable agreement. He reports two favourable cases examined in detail by modern methods.

The principal lesions are then described in connection with the muscles, there being a diffuse and focal increase of the nuclei which are frequently large and polymorphic, considerable atrophy of the muscle parenchyma, and practically negative findings in the neuro-muscular fibres. These changes he regards as occupying the first place in the anatomical picture and forming the essential substratum of the malady.

The author reviews the toxic-infective theories held in regard to mild chorea and Basedow's disease, and similarly on clinical and anatomical grounds regards Parkinson's disease as probably due to a chronic endogenous intoxication whose specific action may be on the muscles.

CHARLES I. LAMBERT.

**THE PATHOLOGY OF FOUR CASES OF EPILEPTIC IDIOCY.**  
(373) HARVEY BAIRD, *Journal of Mental Science*, July 1906, p. 571.

THE author gives the clinical notes and pathological changes found in these cases, and in his conclusions emphasises certain important facts. (1) The gross nature of the lesions in epileptic as

opposed to non-epileptic idiocy. (2) The frequency of meningeal changes, especially of the pia-arachnoid, the constant opacity of which leads him to consider meningitis the primary cause of epileptic idiocy in many cases. Three of the cases recorded are supposed to have been due primarily to meningitis in early but post-natal life, if one can argue from the complexity of the convolutions; in the remaining case the absence of structures in the region of the corpus callosum and of sulci in the left frontal lobe suggests an ante-natal lesion. (3) The larger pyramidal cells appear to be the last to degenerate. DAVID ORR.

### CLINICAL NEUROLOGY.

**RHEUMATIC POLYNEURITIS OF CRANIAL NERVES.** (Sulla (374) polineurite reumatica dei nervi cranici.) FORLI, *Riv. Sper. di Fren.*, Vol. xxxii., Fasc. 1-2.

THE condition of rheumatic paralysis of cranial nerves has been recognised for a long time, but no agreement as to its pathogenesis has been reached. It has been considered by some to be a neurosis, generally hereditary, by others to be secondary to affections of the ears, by others a real infective neuritis; but only recently has the influence of cold been appreciated.

Moebius suggests that it is an infective process determined usually under the influence of cold. Von Sarbo denies the influence of cold and of heredity, and attributes the condition to an exposure of the nerves, due to some anatomical peculiarity of the foramina through which they pass.

The author describes a case of a railway employée who had been much exposed to rain and wind, and who developed a lesion of his 3, 4, 6, 7, 8, and 12 right nerves. The affection came on rapidly and was accompanied by general malaise, pain, and some paresis. Syphilis was excluded. The patient recovered rapidly under antirheumatic treatment. This case was considered to be a case of multiple neuritis of cranial nerves of rheumatic origin, due to some infective process developing under the influence of cold, and attacking the nerves.

R. G. ROWS.

**PERIPHERAL FACIAL PALSY, WITH AUTOPSY.** (Paralysie (375) faciale périphérique : autopsie.) MIRALLÉE, *Soc. de Neurol. de Paris*, July 5, 1906.

THE patient was an arterio-sclerotic, who developed a typical right peripheral facial palsy as the result of a chill, and died rather less

than six weeks later of pulmonary congestion, the palsy being still well marked.

Microscopical examination of the nerve trunk revealed (Marchi) an intense parenchymatous neuritis, more marked peripherally than centrally.

In the corresponding nucleus in the pons (Nissl) the great majority of the cells were swollen and rounded, with obliteration of the protoplasmic prolongations; the nuclei of the cells were distinctly visible and still central, and round them was a pronounced chromatolysis. The left seventh nucleus was absolutely normal, as was the nucleus of the sixth on the right side. The cellular lesion was probably due to a *réaction à distance*, and the case may be taken to indicate that no fibres join the facial nerve from the sixth nucleus.

S. A. K. WILSON.

**THE NEURITIC TYPE OF PROGRESSIVE MUSCULAR ATROPHY.**

(376) **A CASE WITH MARKED HEREDITY.** CHURCH, *Journ. of Nervous and Ment. Dis.*, July 1906.

THE writer first gives a *résumé* of the leading symptoms of this disease as stated by Sainton, who collected and scrutinised in a thesis all the cases published up to 1899. These include a commonly hereditary nature, commencement usually in the lower extremities, paralysis of flaccid type not extending above the lower third of the thigh nor above the proximal part of the forearm, fibrillary tremors, and frequent disturbances of sensation. Post-mortem, there are found interstitial neuritis, degeneration of the posterior columns of the pyramidal tracts, of the cells in the anterior horn, and of those in Clarke's column.

Other cases appearing since those of Sainton are quoted with references.

Full details are given by the writer of a new case which affords a most notable instance of heredity, the disease being certainly present in five successive generations, perhaps apparent in the sixth, and, according to hearsay, in three prior generations—nine in all.

JOHN D. COMRIE.

**TABES, DURING THE DEVELOPMENT OF WHICH APPEARED**  
**(377) A CHANCER, APPARENTLY SYPHILITIC. SLOWNESS**  
**IN ANATOMICAL DEVELOPMENT OF THE MEDULLARY**  
**LESIONS. INTENSE PERIPHERAL NEURITIS IN CON-**  
**NECTION WITH AN ARTHROPATHY OF THE KNEE**  
 VERGER and DE CARDENAL, *Revue Neurologique*, July 15, 1906.

THE name indicates sufficiently the scope of the case recorded and the observations upon it. The writers give references to four similar cases in which a chancre appeared during the course of tabes.  
 JOHN D. COMBIE.

**THE PHYSIOLOGY OF THE LARYNGEAL CRISES OF TABES.**  
**(378) (Physiologie des crises laryngées des tabétiques.) MAURICE**  
**FAURE (Congrès de Lille), *Revue Neurologique*, Aug. 30, 1906,**  
 p. 776.

WHEN a particle of mucus passes, in a normal individual, from the trachea into the larynx, its presence produces a special effect on the laryngeal mucosa, and a reflex immediately results in the shape of a cough or an expiratory "ahem!"

For the tabetic, this succession of sensory and motor phenomena is interfered with: 1st, because the laryngeal mucous membrane is hyperæsthetic or hypoæsthetic; 2nd, because the expulsion reflex is disordered; 3rd, because there is inco-ordination of the muscles of respiration.

For these reasons the relaxed diaphragm may be drawn into the thorax just when it is being expanded by the muscles of inspiration, or conversely the diaphragm may descend when the thoracic cage is falling in; in each case there is no change in the volume and the expiratory effort is annulled. Or the action of the glottis may be so irregular as to prevent an expiratory column of air from producing its effect. It is common to observe the reflex cough determine a series of short, staccato expirations, which have not been preceded by such inspirations, as will fill the thorax with air, and are therefore useless.

As a result, the paroxysmal tickling continues, the patient gets excited, becomes pale and cyanosed, and runs serious risks.

In other cases the whole disturbance is referable to inco-ordination of laryngeal muscles in association with anæsthesia of the larynx. It is obvious that the pathogenesis of these conditions is entirely analogous to what obtains when bladder and rectum are affected. Actual paresis or paralysis of the muscles of the larynx, from peripheral neuritis or from alteration of nuclear cells, may occur, but this is very infrequent.

Muscular spasms in tabes are met with only exceptionally ; they are brief, harmless, and transient. To designate the condition above described as one of laryngeal spasm, is certainly inaccurate. Inco-ordination, relaxation, atony, abolition of reflexes do not characterise spasm. Faure advocates the application of the method of systematic re-education to the larynx and thorax.

S. A. K. WILSON.

**PARALYSIS BY COMPRESSION OF THE PYRAMIDAL TRACT  
(379) WITHOUT SECONDARY DEGENERATION.** (*De la paralysie par compression du faisceau pyramidal, sans dégénération secondaire.*) BABINSKI, *Revue Neurologique (Soc. de Neurol. de Paris)*, July 5, 1906.

IN this case, reported at length by Babinski, the patient was a man of fifty-two, who was under continuous observation for two years previous to his death. His earliest symptoms were slight difficulty and confusion when speaking, and slight paresis of the right half of the body. A diagnosis of intracranial vascular disease with thrombosis was made. Three months later he developed several attacks of Jacksonian epilepsy, beginning in the muscles of the right face, and spreading to the arm and leg. The difficulty in speech had increased, and the paresis also, resulting in a mild degree of contracture. There was no headache whatever, no sickness, no optic neuritis. The cutaneous and tendon reflexes were normal, and equal on the two sides. The diagnosis was reconsidered, but a cerebral tumour seemed unlikely, yet more probable than an hysterical hemiplegia ; in any case, a course of thorough antisiphilic treatment was commenced.

Seven months from the onset of the disease the patient's condition was obviously worse, but the reflexes remained normal and the cardinal symptoms of intracranial neoplasm were absent. An operation, however, was suggested, but refused. At the end of a year and a half the contracture and weakness were much greater, the aphasia was more pronounced, but there was no other change, except double papillitis and slight diminution of visual acuity.

At length, after two years, the patient was operated on, and a tumour weighing 310 grammes was found attached to the dura mater over the left psycho-motor area, compressing but not invading the cerebral substance. Its enucleation was performed with the utmost facility, but unfortunately the patient succumbed immediately after the operation. Examination showed the tumour to be a myxo-sarcoma.

From beginning to end of the illness there was neither headache nor vomiting, and the reflexes were absolutely normal. Babinski thinks that compression, as opposed to invasion, of the

cerebral cortex produces a clinical picture such as has been detailed. He ventures to describe the condition as one of "pseudo-hysterical organic paralysis," admitting that in this particular case there was no intrinsic sign whereby he could distinguish between functional and organic disease; extrinsic signs were requisitioned to establish the diagnosis.

S. A. K. WILSON.

**TWO CASES OF TUMOUR OF THE CORPUS CALLOSUM.** (*Deux cas (380) de tumeur du corps calleux, avec autopsie.*) RAYMOND (Congrès de Lille), *Revue Neurologique*, Aug. 30, 1906, p. 772.

THE first patient had a stroke three months before his death. Immediately after the ictus a general deterioration of his mental faculties was remarked, in addition to a progressive hemiplegia and hemitremor, and subsequently a hypoesthesia of the same side. At the autopsy a large vascular gliomatous tumour was found in the anterior two-thirds of the corpus callosum, spreading into the frontal lobe anteriorly and laterally into the left centrum ovale. In addition, a second focus of disease was discovered in the ascending parietal convolution of the left side. In that hemisphere the tangential fibres of Exner were atrophied to a considerable extent, and the superior longitudinal bundle, the occipito-frontal tract, and the cingulum were destroyed.

The second patient did not present any symptom for eight months beyond special mental disorders; two months before his death he developed a progressive left hemiparesis, with great ataxia and anaesthesia on the same side, as well as a left homonymous hemianopia. A diagnosis was made of tumour of the corpus callosum spreading into the right optic thalamus, and at the sectio the posterior third of the corpus callosum was found to be invaded by a highly vascular sarcoma, which had made its way into the posterior two-thirds of the right optic thalamus, and the posterior third of the internal capsule, destroying the thalamic radiations and the inferior longitudinal fasciculus as well.

The special mental syndrome which the author seeks to attach to lesions of the corpus callosum consists of eccentricity in manner and in actions, hiatuses in memory, want of sequence in ideas, coupled with an apparent conservation of the intelligence.

S. A. K. WILSON.

**THE BORDERLAND OF EPILEPSY.** Sir WILLIAM GOWERS, (381) *British Medical Journal*, July 7, 1906, p. 7.

IN this, the first of two lectures, the author deals more especially with the subject of vertigo. He insists that epilepsy cannot be

sharply marked off from functional diseases, and that there is a borderland of diseases which fades in either direction.

Of the different varieties of vertigo, the only one that in practice is liable to be confounded with epilepsy is labyrinthine vertigo. In minor epilepsy the subjective, objective, and motor vertigo are all in the same direction, and are probably due to inequality of discharge in the two hemispheres. In aural vertigo the subjective and objective rotation are usually in different directions. Both affections have a sudden onset, and both may have a brief duration, though this is rarer in the case of aural vertigo. The author lays stress on the fact that consciousness may be not merely blurred, but momentarily lost, in aural vertigo, and quotes cases in support of this statement. Again, sight may be lost in this affection for a few seconds, when consciousness is retained, still further increasing the resemblance between epileptoid forms of aural vertigo and minor epilepsy. Another symptom that is frequently mistaken for an epileptic aura is the sense of impulsion felt by some patients at the onset of a vertiginous attack. Although in epilepsy there may be a sense of involuntary movement, there is never the feeling of being hurled or even struck to the ground, as by an outside force, that is found in the other cases; when this occurs it is characteristic of aural vertigo.

ERNEST JONES.

**LECTURES ON THE BORDERLAND OF EPILEPSY. II. VERTIGO.**

(382) Sir WILLIAM GOWERS, *Brit. Med. Journal*, July 21, 1906, ii., p. 128.

IN the first lecture the writer had considered the features of aural vertigo that resembled epileptic symptoms—suddenness, brevity, loss of consciousness, loss of sight, and the curious sensation of impulsion downwards as by an external force. In addition, other cephalic sensations may cause difficulty in diagnosis. In one case a sudden sensation would appear in one side of the head, seeming as if it would throw the patient down. In another a sudden sensation would dart from the back of the neck over all the head, with a strong sense of impending loss of consciousness. In yet another there was a feeling of a sudden "rush" to the head, and that consciousness would be lost unless the patient at once sat up. The relation of these attacks to posture, and their association with tinnitus, is most significant of aural vertigo, as otherwise they would often suggest epilepsy. In some instances the attack is induced in the sitting posture, and relieved by lying down; in others the reverse occurs; and sometimes lying on a certain side is the factor concerned.



Another point of resemblance is that aural vertigo, like epilepsy, may occur during sleep. This fact has no doubt to do with the alteration in pressure that must occur in the semicircular canals during the supine posture. When visual and muscular influences are cut off from the equilibril centre, as they are during sleep, impulses still reach it from the semicircular canals, and the combination of sleep and posture may effect what posture alone cannot. In this connection the author proffers his opinion that the sensation of falling through space, so common during sleep, may be due to contraction of the stapedius, which lowers the pressure in the canals.

The cephalic sensations just referred to are not the only ones that may give rise to difficulty in the diagnosis, for they may be reflected and appear as curious feelings elsewhere in the body, particularly at the heart. It is also important to remember that an epileptic seizure may be ushered in by labyrinthine giddiness, tinnitus, and vertigo; still such tinnitus is not pronounced or persistent.

Aural vertigo and epilepsy, neither of which is a rare disease, may coincide in the same patient; in addition, the one sometimes seems to exert an influence on the other.

A more difficult group to classify is what the writer calls pseudo-aural vertigo. In this, symptoms of labyrinthine vertigo, of the character seen in no affection except ear disease, exist without tinnitus or any evidence of organic labyrinthine disease. The pathology of this group is at present very mysterious.

In the treatment of labyrinthine vertigo the combination of bromides with gelsemium and hyoscine is suggested.

ERNEST JONES.

**EPILEPSY WITH UNILATERAL MANIFESTATIONS.** (*Epilepsie* (383) *mit Halbseitenercheinungen.*) BRATZ and LEUBUSCHER, *Neurol. Centralbl.*, Aug. 16, 1906, p. 738.

THE authors refer to recent writers who support the view of the organic nature of epilepsy. Heilbronner has shown that the speech centre is the originating focus of some cases of epilepsy with speech defects. Redlich has lately revived the old hypothesis that idiocy, cerebral palsies of children, and early epilepsy are due to the same pathological process; this view was based on the numerous cases of early epilepsy that show either a slight hemiparesis or asymmetry in the tendon reflexes. The authors have had many cases with one-sided symptoms, and at the autopsy changes have been found in the contralateral Ammon's horn. An example of this is quoted in detail. The patient was admitted thirteen years ago, when aged twenty-three. But for one attack in his seventh

year, he was quite healthy till sixteen; after this he had fits weekly. On admission he showed some deviation of the tongue to the right; five years later it was noted that he fell towards the left in his attacks. Mental deterioration was rapid and progressive. Three days before death it was noticed that in a fit his eyes were turned strongly to the left, although, in a stuporose state before the fit, they were turned to the right. Clonic movements occurred much more on the right side than on the left. At the autopsy the left Ammon's horn was much shrunken; there were no other changes. During life the diagnosis lay between hysteria, epilepsy, and organic brain disease, but was made correctly. The authors state that bilateral changes in Ammon's horn are rarely found except in cases of epilepsy, combined with idiocy. The origin of these changes is probably to be attributed to foetal trauma or infection.

ERNEST JONES.

**HEART-NEUROSES AND BASEDOW'S DISEASE.** (*Herzneurosen* (384) *und Basedow.*) FISCHER, *Muench. med. Wchnschr.*, Aug. 7, 1906.

THE writer refers in the first place to the subject of removal of the thyroid in the treatment of the disease, and recommends the removal at most of one-half of the gland to begin with. He recommends, instead of operation, the antithyroid preparation "Rodagen" (derived from the desiccated milk of goats from which the thyroid gland has been removed).

The paper deals chiefly with cases corresponding to the "formes frustes," which the writer believes are much more common than is generally supposed. Thus cases diagnosed as "neurasthenia," "anæmia," or "heart-neurosis," particularly in women suffering from menstrual disorders, he considers frequently are of the nature of imperfectly developed Basedow's disease. The chief complaint is always irregularity or pain in the heart, with shortness of breath or faintness. If tonic treatment fails in these cases, he recommends the trial of two grammes of rodagen thrice daily, combined with rest. He states that he has several times seen decrease of the enlarged thyroid under this treatment, notably in one case where the goitre had lasted over twenty years, and distinctly diminished after six weeks' treatment.

JOHN D. COMBIE.

**THE PROGNOSIS OF TETANY IN ADULTS.** (*Die Prognose der*  
(385) *Tetanie der Erwachsenen.*) L. V. FRANKL-HOCHWART, *Neurol.*  
*Centralbl.*, July 16, 1906, p. 642, and Aug. 1, p. 694.

THE prognosis of this affection is usually considered to be very favourable; thus, of 264 cases of whom the author had hospital notes, 215 were discharged as cured, 42 improved, and 6 unimproved. Extensive experience had suggested, however, to the author that this opinion might be unduly favourable, owing to the fact that it leaves out of consideration the tendency of the affection to recur. He remarks how rarely such problems are revised *de novo* in neurology, but determined so to do in this instance. He made observations of 160 patients in the past twenty years, and, by holding a recent inquiry, was able to follow the after-history of 55 of these. The tetania strumipriva and the stomach tetany was excluded, as our knowledge seems to be on more secure ground in the case of these. The present cases refer to the endemic tetany of cobblers and tailors, puerperal tetany, and that due to acute infectious disease.

The 55 cases, of which an account is given, are divided into five groups. Group I. includes 7 cases of chronic or repeatedly recurring tetany. Group II. includes 19 cases in which spasms were replaced by paræsthesiæ or cramp feelings. Group III. also includes 19 cases, in which there were no spasms present, and which had symptoms reminding one of myxœdema. Group IV. includes 11 fatal cases, death being due to other diseases. Group V. includes 9 cases that recovered. These groups are analysed with respect to the variety and situation of the tetany present, and the frequency with which the symptomatic triad (of Trousseau, Erb, and Chorstek) occurred. The results of the inquiry are as follows:—Of the 55 cases, 11 had died at a relatively early age; of the 44 living, 9 were healthy—about 20 per cent. Over four-fifths of the patients were more or less permanent sufferers. The prognosis as to recovery is thus very considerably worse than is usually thought.

ERNEST JONES.

**ANOTHER CASE OF INTERMITTENT CLAUDICATION.** (*Ein*  
(386) *weiterer Fall von angiosklerotischer Bewegungsstörungen des*  
*Arms.*) W. ERB, *Deut. Zeit. f. Nervenheilk.*, Bd. 30, 1906,  
p. 201. See this Review, Feb. 1906, p. 153.

THE patient was a woman of 57 with diabetes mellitus of two years' standing. For about one year she had had smarting pains in both legs and arms, then some swelling of the legs. In the right arm there was great smarting, and a feeling as if the right hand were thicker than the other and unable to be closed properly.

There was no visible swelling. After using it much the hand became somewhat purplish in colour and very feeble, with great pain in the upper arm. These symptoms passed off on resting. On examination the heart was found to be hypertrophied, there was marked arterio-sclerosis; the right radial pulse was completely absent, although the artery was not especially hard. The brachial pulse was just palpable, and the artery was very wiry. Pulsation in the right subclavian was quite good. Blood pressure on the right 70, on the left 130 mm. Hg. Erb considers the case as one of typical Dyskinesia angiosklerotica brachii.

J. H. HARVEY PIRIE.

**TWO CASES OF BI-TEMPORAL HEMIANOPIA.** (*Deux cas d'hémianopsie bitemporale.*) GALEZOWSKI, *Soc. de Neurol. de Paris*, July 5, 1906.

Two cases of bi-temporal hemianopia in middle-aged women, of gradual onset, with pallor of the optic discs and considerable diminution of visual acuity. Galezowski gives his reasons for attributing the defect to hypertrophy of the pituitary body, possibly neoplastic in origin.

S. A. K. WILSON.

**BLINDNESS OF CORTICAL ORIGIN, FROM DOUBLE HEMIANOPIA.** (*Cécité corticale par double hémianopsie.*) RAYMOND, LEJONNE, and GALEZOWSKI, *Soc. de Neurol. de Paris*, July 5, 1906.

THE patient was an arterio-sclerotic, fifty-seven years of age. On December 3, 1905, he had a sudden attack of giddiness and vomiting, and seemed to be impelled towards the left. Seven days later he had another attack of giddiness, and felt himself forced towards the right. On December 25 he suddenly felt a tingling of the whole of his left arm, and a moment later he became completely blind. On January 4 he began to distinguish objects again, but on the 6th he had another attack, and remained more or less comatose till the end of the month. As a result he became absolutely blind again, but by April he had once more begun to distinguish objects faintly.

On examination, the field of vision was entirely gone, except for a minute area round the fixation point in each eye. In this zone his visual acuity was  $\frac{6}{12}$ . There was no loss of colour vision; pupil reactions were normal; and the fundus showed no abnormality in either eye. A consideration of all the symptoms makes

it probable that after the first attack there had been hemianopia, which did not involve the fixation point, and remained unnoticed. Whenever a lesion occurred in the other occipital lobe, however, the cortical centre for macular vision was partially involved, and the blindness became complete. As in a number of published cases, central vision gradually returned, though not quite to the normal.

S. A. K. WILSON.

**THE ANTAGONISM OF THE CUTANEOUS AND THE TENDON**  
(389) **REFLEXES IN SPASTIC PARAPLEGIA.** (Étude sur l'antagonisme des réflexes cutanés et tendineux dans les paraplégies spasmodiques.) NOICA and MARBE, *Soc. de Neurol. de Paris*, July 5, 1906.

THIRTY-EIGHT cases of spastic paraplegia, of various types, were examined.

In five cases the cutaneous reflexes were normal. In twelve, some of the cutaneous reflexes were present, others diminished or absent. To quote one instance: the upper and middle abdominal reflexes were intact, the lower abdominal and the cremasteric were absent, the anal reflex could still be elicited, but the gluteal was not obtained.

In two cases all the cutaneous reflexes were abolished. Evidently the principle of antagonism is not absolute.

S. A. K. WILSON.

**REMARKS ON THE MEANING AND MECHANISM OF**  
(390) **VISCERAL PAIN.** MACKENZIE, *Brit. Med. Journ.*, June 30, 1906.

THIS, the last of a series of three papers, deals with the mechanism by which visceral pain is produced. The writer refers to the three tissues by which pain is exquisitely felt in the body wall, viz., the skin, the muscular layer, and particularly the loose cellular tissue immediately outside of the peritoneum. With regard to the opinion of some that the viscera are incapable not only of originating sensations from stimuli, such as cutting, tearing, or burning, which affect the skin, but even of producing any sense of pain directly, he adduces the familiar example of a large enema, which serves to produce severe griping pain, accompanying muscular contraction of the rectum. This pain originates undeniably in the rectum, but is referred in most people to the region immediately above the pubis.

This peculiarity of visceral pain in being "referred," he

- explains as follows: Constant afferent impulses pass from the viscera to the central nervous system, but set up no sensations; if, however, a morbid process in a viscus gives rise to exaggerated impulses, these affect neighbouring centres in the spinal cord, and so sensory, motor, and other nerves connected with this region of the cord are stimulated. According to the writer, therefore, visceral pain is of the nature of a viscerosensory reflex, while the various muscular contractions set up in visceral disease correspond to visceromotor reflexes.

Angina pectoris is taken as an example, and the writer explains its symptoms by reference to his theory. Thus the pain experienced over the region of the heart and down the arm is not a direct pain in the heart, but a referred pain in the distribution of the upper dorsal nerves, which are connected with the splanchnic nerves of the heart. Similarly the agonising feeling as if of the breast-bone snapping, or of the chest being squeezed in a vice, is explained as a visceromotor reflex, in which the intercostal muscles are thrown into a state of extreme spasm.

JOHN D. COMRIE.

**ON THE THEORETIC AND PRACTICAL MEANING OF HEAD'S  
(391) ZONES IN DISORDERS OF THE DIGESTIVE ORGANS.**

(Zur theoretischen und praktischen Bedeutung Head'scher Zonen bei Erkrankung der Verdauungsorgane.) KAST, *Berl. klin. Wchnschr.*, July 30, 1906, and August 6, 1906.

IN two papers the writer discusses the clinical value which can be assigned to Head's zones of cutaneous hyperæsthesia in connection with disease of deep-seated organs, after he has examined some two hundred cases of disease of the abdominal and thoracic organs with reference to the point.

He found that in many cases the hyperæsthesia affected not only the senses of pain and temperature, as Head found, but also the sense of touch. In all cases, however, the minimal stimulus necessary to produce a sensation was the same over the zone as on the rest of the skin, although the sensation provoked differed in acuteness. In explanation of this peculiarity, he offers the analogy that each segment with its splanchnic and spinal nerves is tuned to a certain pitch; the constant reception in the spinal cord of stimuli from diseased organs, though these are not felt as pain, produces an effect comparable to loss of pitch, and hence the change in sensibility. This circumscribed defect in a segment he regards as a local neurasthenia.

He discusses at some length the question as to whether sensations of any sort are produced by affections of internal

organs, but finally dismisses as untenable the opinion of Lenander, founded upon his experience of operations done under local anæsthesia, that the organs innervated by the vagus and sympathetic nerves are devoid of sensation, except in so far as damage to and stretching of the parietal peritoneum are concerned, or in cases where an inflammatory process spreads from internal organs to neighbouring spinal nerves. He believes that the hyperæsthesia of Head's zones demonstrates that the sensations of pain originate in definite organs.

The presence of a hyperæsthetic zone does not necessarily follow even serious disease of the corresponding organ. Thus in serious gastric ulcer he repeatedly found the zone absent. Again, out of twelve cases of gastric cancer only one showed Head's zone. The writer does not agree with the suggestion of Willoughby that the position of an ulcer in the stomach can be determined by the position of the superficial zone affected. Though the zones are usually discoverable in appendicitis, they are of little diagnostic value, since the same areas are affected in catarrh of the small intestine. On the other hand, the writer believes that the level of the zones affected is of great value in the indication of the particular organ at fault when general symptoms like vomiting and cardiac weakness only are present.

A word of warning is given against mistaking for Head's zones the sensitive areas of skin found in emaciated individuals where the skin is stretched over the rib margins and iliac crests.

On the whole, the writer does not attach great value to these zones as a practical method of clinical examination.

JOHN D. COMRIE.

#### PROJECTION OF PAIN SENSATION FROM THE LOWER TO THE

(392) **UPPER EXTREMITY.** (Ueber Projektion des Schmerzempfindung von der unteren auf die obere Extremität bei Herd im Dorsalmark.) M. LEWANDOWSKY, *Centralbl. f. Nervenheilk. und Psychiat.*, No. 218, Aug. 1, 1906, p. 593.

THE case was that of a young girl with symptoms of localised compression of the spinal cord. Operation revealed a sarcoma of the vertebræ, invading the dura, and the sectio 4½ months later showed a complete transverse softening of the cord at the level of the 4th dorsal segment.

In the interval there was complete flaccid paralysis of the lower half of the body. The left leg was quite anæsthetic and the reflexes lost; in the right there was a little sensation and very feeble reflexes. Strong faradisation of the right leg caused no pain, but a current which a healthy person could scarcely bear when

applied to the left leg for about two minutes then caused severe pain. But more remarkable than the delayed transmission was the projection of the pain. It was referred usually to the left arm, occasionally to the operation wound, but never to the right arm or any part of the body innervated from below the 4th dorsal segment. The pain lasted a few seconds, but once elicited, further stimuli of a few seconds' duration sufficed to cause it. No other form of stimulation had a like effect.

There must either have been a "track-jumping" in the cord or (which the author thinks more probable) conduction to the cortex by a remnant of path coming from the lower extremity, and false projection outwards from the cortex. J. H. HARVEY PIRIE.

**OBSERVATIONS ON THE SO-CALLED "TACTILE APHASIA."**

(393) (*Considerations sur la soi-disant "aphasie tactile."*) DEJERINE, *Revue Neurologique*, July 15, 1906.

THE writer records a case with symptoms similar to those of one regarded by Raymond and Egger as a new type of aphasia and named by them "tactile aphasia." Briefly these consist in the fact that the patient though able to describe generally the size, shape, consistence, and temperature of objects placed in one hand, is incapable of telling their name. This condition, known as "tactile paralysis," was described by Wernicke in 1896, and the writer protests against the name of tactile aphasia as a misnomer. These cases owe their failure to name objects to defective information brought to the cerebral centres by the sensory nerves of the affected hand; while the fact that there is no aphasia is shown by the ready naming of all objects placed in the sound hand. In all cases of real aphasia, motor or sensory, according to the writer, the aphasic person preserves the images of objects in his mind, but has lost their names. It would therefore be as reasonable, he says, to call deafness auditory aphasia, or loss of taste gustatory aphasia, as to apply the name of "tactile aphasia" to such cases. The illustrative case in the paper is described in full detail.

JOHN D. COMRIE.

**CEREBELLAR ASYNERGY AND INERTIA.** (*Asynergie et inertie*

(394) *cérébelleuse.*) BABINSKI, *Soc. de Neurol. de Paris*, July 5, 1906.

THE characteristic features of cerebellar disease are titubation, intention tremor, scanning speech, asynergy, disturbance of kinetic equilibrium and of diadococinesis, and catalepsy. All these phenomena may be connected one with the other if we regard



the cerebellar mechanism as having two functions, one destined to insure synergy of movements whose combination constitutes a volitional act, and the other intended to combat inertia.

Thus intention tremor may be explained by a lack of co-ordination among the various elementary movements of the upper limb, in particular by unharmonious functioning of antagonistic muscles. The tremor, further, is sometimes associated with abrupt movements, movements *sans mesure*. Thus the patient can bring his hand up to his mouth briskly, but he is usually unable to arrest the movement just when he wants to; in other words, he is unable to control the inertia of a moving body.

Again, diadococinesis—the function which permits us to execute rapidly a succession of volitional movements—consists in an association of exciting and restraining action, and is intended to overcome the inertia of the motor apparatus alternately at rest and in motion. The disturbances of equilibrium are due to the abolition of diadococinesis, and the scanning speech may legitimately be attributed to inertia.

S. A. K. WILSON.

## PSYCHIATRY.

### ON KORSAKOW'S SYMPTOM-COMPLEX IN BRAIN SYPHILIS.

(395) (*Ueber den Korsakow'schen Symptomencomplex bei Hirnlues.*)

L. ROEMHELD (of Horneegg), *Arch. f. Psych.*, Bd. 41, H. 2.

PATIENT was a woman at the climacterium who, in the spring of 1904, became forgetful and began to fabricate; she had headache at night, suffered from attacks of dizziness and transitory mental disturbance, and was apathetic; her gait became unsteady. On admission (July 1905), there was pain on pressure over the left side of the skull; pupils were small and sluggish in reaction; the right side of the face was weak and showed movements of the nature of a tic, which sometimes even spread to the right arm and leg. Mentally patient was very apathetic; speech was slow and hesitating; she was quite disoriented, and had exceedingly poor retention. In hospital patient had several attacks of fainting and dizziness; she fabricated with the greatest freedom; the pupils, which had at first acted sluggishly, after some time became quite fixed; examination of retina showed choked disc. Treatment by mercury and by iodide separately was ineffective, but mixed treatment with injections of iodipin was successful, and patient in two months made an excellent recovery. On discharge (Nov. 1905) she showed no mental defect, but had an amnesia for the period of her illness. She still occasionally complained of headache; there was no longer inequality of facial innervation; the knee-

jerks were equal and the sign of Romberg absent. The eye still showed choked disc.

Several months later, patient returned on account of attacks of dizziness, which again yielded to the same treatment. The author discusses the question whether the cause is to be looked for in the vascular changes, or in a gumma with increased intercranial pressure, or in the syphilitic intoxication acting in a manner analogous to chronic alcoholic poisoning.

C. MACFIE CAMPBELL.

**ON ATYPICAL ALCOHOLIC PSYCHOSES. A CONTRIBUTION (396) TO THE KNOWLEDGE OF THE HALLUCINATORY DEMENTIA OF THE ALCOHOLIC, AND OF ALCOHOLIC PSEUDO-PARALYSIS.** (Ueber atypische Alkoholpsychosen. Beitrag zur Kenntniss des hallucinatorischen Schwachsinnns der Trinker und der alkoholistischen Pseudoparalyse.) F. CHOTZEN (of Breslau), *Arch. f. Psych.*, Bd. 41, H. 2, pp. 383-481.

IN this clinical contribution to the subject of the alcoholic psychoses the author shows the necessity for defining more clearly the grounds upon which certain psychoses are called alcoholic, and the difficulty of differentiating these psychoses from others of non-alcoholic origin. The difficulty is greatest with various chronic conditions, but even in the acute conditions the symptomatological differentiation is by no means easy. The acute hallucinatory condition arising on an alcoholic basis—the “Acute Hallucinosis” of the Germans—is in its typical form a well-defined picture: auditory hallucinations dominate this picture, hallucinations of smell and taste are absent, there are no hypochondriacal complaints, and the outcome is recovery. Such a condition, however, may arise without alcohol being an ætiological factor; on the other hand the symptom-complex, even on an alcoholic basis, may be complicated on the one hand by hypochondriacal symptoms without the outcome being different, on the other hand by motor symptoms, even by marked stupor. Several attacks of acute alcoholic psychoses may be followed by a chronic psychosis presenting close similarity with chronic non-alcoholic psychoses. In addition to the fact that the ætiology, even of the well-defined alcoholic psychoses, is complicated and obscure, the symptomatological likeness to other psychoses makes the differentiation extremely difficult. The acute hallucinatory psychosis on an alcoholic basis is difficult to separate from hallucinatory episodes belonging to Dementia Præcox. The fact that a chronic paranoic psychosis has been preceded by such an acute hallucinatory con-

dition does not warrant us in calling it alcoholic paranoia, unless the chronic psychosis presents in itself distinctive features; the hallucinatory onset may be looked on merely as an episode in a Dementia Præcox development.

The author cites four cases to show the heterogeneous nature of this group of paranoic conditions of alcoholic origin, and the difficulty in determining the ætiological factors.

He then passes to the subject of the hallucinatory dementia of the alcoholic, and reports various observations. He starts from Kraepelin's description and shows what a variety of cases come under this head, and how other elements than the alcoholic factor play an important part. Even the initial deliria which precede one large group are atypical; the psychoses vary in their symptomatology and frequently show degenerative phenomena. Cases belonging to another group are progressive with fantastic ideas of greatness and of being influenced, but even in these cases the course and outcome vary; some are closely related to Korsakow's psychosis. In this group there is not the definite deterioration of the previous group; recovery is possible.

Alcoholic pseudo-paralysis is not necessarily a combination of this hallucinatory dementia with the Korsakow symptom-complex. The picture includes, in addition to the mental symptoms, cerebral focal symptoms due to hæmorrhages and parenchymatous degeneration. Vascular changes and toxic influences are both important elements. The various stages in the development of alcoholic pseudo-paralysis are closely analogous to conditions met with in the group of infection psychoses, *e.g.* stuporous conditions, and conditions of confusion with motor symptoms. In this context the author refers to a case of a markedly catatonic psychosis in association with focal brain symptoms of alcoholic origin with memory and retention disorder. Korsakow's psychosis may result in a characteristic terminal condition of paranoid nature, with the delusions limited to the idea of annoyance by those forming the environment of the patient.

To sum up: alcoholic pseudo-paralysis may arise from the association of arterio-sclerosis with alcoholic psychoses; secondly, it may develop from the association of the polyneuritic complex or cerebral focal symptoms with either a late stage of a typical Korsakow's psychosis or with other forms related to the infection psychoses, or to forms of organic vascular brain disease; thirdly, it may arise from the association of alcoholic focal brain symptoms with a degenerative megalomaniac psychosis, or with "mania."

C. MACFIE CAMPBELL.

**MANIC-DEPRESSIVE INSANITY AND ARTERIOSCLEROSIS.**

(397) (*Manisch-depressives Irresein und Arteriosklerose.*) ALBRECHT,  
*Allg. Ztsch. f. Psych.*, Bd. 63, H. 3 und 4.

THE writer reports briefly 54 cases of manic-depressive insanity, in 18 of which (*i.e.* 30%) arteriosclerosis was present. This ratio is considered high because only 40% of the alcoholics showed the signs of arteriosclerosis. Dementia præcox gave 10%. Kraepelin has mentioned the early occurrence of vessel sclerosis in manic-depressive insanity, and considers that the prognosis is thereby rendered less favourable. The investigation led to the following conclusions.

General arteriosclerosis is more frequent and appears earlier in manic-depressive insanity than in the other non-toxic psychoses or in those unaccompanied by period fluctuations in the affect.

The causal relation of the vessel disease and manic-depressive insanity is a double one. In a majority of cases the mental disorder with its emotionally conditioned variations in blood pressure and resulting anomalies of nutrition in the vessel walls, creates an especially favourable ground for the development of arteriosclerosis.

In a minority of cases the senium, with its accompanying arteriosclerosis, especially in hereditarily burdened individuals, furnishes the final conditions for the outbreak of manic-depressive insanity.

G. H. KIRBY.

**THE HEBEPHRENIC FORMS OF DEMENTIA PRÆCOX. (Ueber**

(398) *die hebephrenischen Formen der Dementia praecox Kraepelins.*)  
 KLIPSTEIN, *Allg. Zeitschrift f. Psych.*, Bd. 63, H. 3 und 4,  
 p. 512.

THE hebephrenia of Kraepelin comprises those forms of dementia præcox in which the mental weakness develops either gradually or with the signs of a sub-acute, rarely acute mental disturbance; to be excluded are those forms with well-marked catatonic states, as well as those in which there develop with retention of clearness prominent delusions, which remain for years in the foreground.

The author's observations are based on 100 cases; no clinical histories are furnished. In a majority the onset was between the fifteenth and twenty-fifth year. The symptomatology of the chronic sub-acute and acute forms is discussed in detail.

The slowly developing cases show from the beginning the signs of dementia and further hallucinations, falsifications of memory, symbolic interpretations and delusions, which are, how-

ever, expressed with slight show of emotion and remain unsystematised. Peculiarity in behaviour and queerness of speech develop rapidly.

The acute and sub-acute cases are divided into (1) simple and (2) compound forms. Under (1) states of excitement and depression are described. In (2) the disturbance of will and feeling are more strongly expressed, hallucinations and delusions are more prominent.

Emotional decay, passivity, reduction in active attention, and defect in concentration are the permanent and fundamental disturbances which appear in all forms of hebephrenia.

The course may be gradual and even, but is more frequently fluctuating with remissions and exacerbations. Recovery without defect has not been observed.

No sharp line exists between the acute and chronic forms, and the acute disturbances can be considered only as phases in a fundamentally chronic disorder.

The acute and sub-acute forms of hebephrenia show numerous transitions to the catatonic variety of dementia præcox, and the chronic forms lead over to the paranoid types of dementia præcox. Kraepelin's attempt to separate the small group of paranoia from dementia præcox cannot be supported.

The writer proposes the following tentative grouping within the department of dementia præcox.

(1) Acute and sub-acute types, with remissions and catatonic symptoms. Here belong Kraepelin's catatonic form, and the acute and sub-acute forms of hebephrenia described by the author.

(2) Chronic types with delusions. Here may be grouped chronic forms of hebephrenia, Kraepelin's paranoid form of dementia præcox, and the small group of paranoia.

G. H. KIRBY.

#### **THE HEBROID PARANOID GROUP OF DEMENTIA PRÆCOX—**

(399) **CLINICAL RELATIONS AND NATURE.** DERCUM (of Philadelphia), *Am. Journ. of Insanity*, April 1906.

It is conceded that the dementia præcox group forms the centre of discussion among the psychiatrists of to-day. Kraepelin, the originator of the group, states that the term implies nothing more than the unfavourable prognosis and the appearance of the disease in youthful age. There is no sharp dividing line between the different types (hebephrenic, catatonic, paranoid), and the existence of a paranoid type is questioned. Certain depressive and expansive phases of dementia præcox resemble similar phases in manic-depressive insanity, but a careful analysis of any given case

is commonly sufficient to make the distinction. Hallucinations, which play such an important rôle in both phases of dementia præcox, play no part whatever in manic-depressive insanity; confusion dominates the dementia præcox picture, but is at most only an incident in the manic-depressive picture. Dementia is commonly understood to be the most important symptom of dementia præcox, but facts do not justify such a position. Dercum makes a comparison between the reduction of a simple senile dementia and dementia præcox. Dementia præcox is a quantitative and not a qualitative change—memory, consciousness, orientation are seriously affected in senile dementia, but are frequently preserved to an astounding degree in even advanced cases of dementia præcox. Confusion, and not dementia, should be regarded as the striking feature of at least the earlier stages of dementia præcox—completely cured cases probably constitute a group by themselves, and should be separated from the great mass of dementias.

The delirium-confusion-stupor complex of such toxines as lead and alcohol is considered, and the possibility of dementia præcox being a toxic disturbance is discussed.

Diem's twelve cases of simple and uncomplicated dementia are referred to—in these cases there were no elements of depression or expansion; no hallucinations, delusions, grimaces, clownism, or stereotypy; these cases should be considered as true primary dementias.

Finally, the author discusses briefly Kraepelin's division of paranoia, and suggests that the first form be designated as heboid paranoia; the second form as hallucinatory paranoia; and the third form as paranoia simplex.

C. H. HOLMES.

**THE TREATMENT OF ACUTE INSANITY IN A GENERAL  
(400) HOSPITAL.** BROWER (of Chicago), *Journ. of Am. Med. Assoc.*,  
July 14, 1906.

THE increase in insanity is due to the feverish activity of the age in which we live; the diminished curability is due to racial degeneracy, and to the difficulty of securing promptly the scientific treatment necessary to aid in restoration. One hundred years ago the insane were considered either as recipients of Divine favour or as victims of diabolical possession. At the present time, while insanity has been raised to the dignity of a genuine sickness, our medical colleges give entirely too little attention to the study of psychiatry and treatment of the insane. The delays incident to the commitment and delivery to hospitals for the insane are often prejudicial to the recovery of these patients.

Brower considers home treatment a failure, but has met with some success during the past thirty years in the treatment of acute cases in the wards of general hospitals. The "autotoxic and exhaustion" cases lend themselves particularly well to this plan. The Weir-Mitchell rest cure is the form of treatment recommended—particular attention being paid to the general constitution of the patient, and rheumatism, gout, syphilis, tuberculosis, etc., being accordingly treated.

C. H. HOLMES.

### TREATMENT.

**ATTEMPTS AT TREATMENT OF CERTAIN CASES OF CONTRACTURES, SPASMS, AND TREMORS OF THE LIMBS BY LOCAL INJECTION OF ALCOHOL INTO THE NERVE TRUNKS.** (*Essais de traitement de certains cas de contractures, spasmes, et tremblements des membres par l'alcoolisation locale des troncs nerveux.*) BRISSAUD, SICARD, TANON, *Revue Neurologique*, July 30, 1906.

ENCOURAGED by the favourable action of deep injections of alcohol in cases of facial neuralgia and facial spasm, the writers were led to try the same remedy in cases where the limbs were affected. They give a reference to previous work on the cranial nerves.

In the first place, they made numerous experiments on the sciatic nerve of dogs and rabbits to test the effect and limit of safety in the injection of alcohol into a mixed nerve. Various strengths of alcohol, from 5 per cent. up to 80 per cent., were tried. After 80 per cent., the dogs sometimes showed a paresis of the foot, which, however, disappeared after one or two days. On histological examination, the sciatic nerve showed, forty-eight hours after the operation, an increased vascularity, and later degeneration of some of the fibres.

Clinically, in man, the writers used alcohol of 80 per cent., containing 1 per cent. of stovaine, injecting about two cubic centimetres high up in the course of the sciatic nerve, which lay at a depth of three to nine centimetres, according to the stoutness of the individual. Immediately after the injection, coldness and anaesthesia of the limb, some paresis of the foot, loss of the Achilles-jerk, and of all spasm or tremor in the limb were the noticeable features. Later, up to ten weeks after a single injection, absence of all clonus and of the Achilles-jerk, but no anaesthesia, paralysis, modification of the electrical reaction, or trophic changes were observed.

Twelve cases are recorded in which the method was used with benefit, viz. spasm of the foot of fifteen years' duration; hemi-

plegia with contracture (two cases); hemiplegia with athetosis (two cases); spastic paraplegia (two cases); paralysis agitans; pseudo-bulbar paralysis with clonus; and sciatica (three cases).

In later researches the writers had satisfactory results with a strength of 40 to 45 per cent. of alcohol, but found that the effect of 20 per cent. lasted only a few days. JOHN D. COMRIE.

#### **CONTRIBUTION TO THE TREATMENT OF BASEDOW'S DIS-**

(402) **EASE WITH RÖNTGEN RAYS.** (*Beitrag zur Behandlung Basedow'scher Krankheit mit Röntgenstrahlen.*) SKŁODOWSKI, *Deut. med. Wchnschr.*, Aug. 16, 1906.

THE writer refers to experiments showing that the application of X-rays have an effect in causing atrophy of the parenchyma of organs to which they are applied, and states that Senn was the first in 1903 to put this to practical use by diminishing the size of a leukæmic spleen. He gives a reference to cases of Basedow's disease treated successfully by Gori through these means. He gives also a reference to a record of five typical cases of Basedow's disease similarly treated by Widermann, in which the exophthalmus and the pulse remained uninfluenced, though the nervous symptoms abated and the weight was increased.

He gives full details of a well-marked case of his own, in which for a month the region of the thyroid gland was exposed one-half on every alternate day from a distance of 20 cm. for ten minutes at a time to the action of X-rays. At the end of this time the nervous symptoms had almost disappeared, though the pulse and exophthalmus remained as before, and the circumference of the neck increased somewhat. The sweating ceased also, and the patient felt well and increased greatly in weight.

References are also given to somewhat similar experiences of von Stegmann and Beck, the latter of whom used the X-rays as after-treatment in operation cases. JOHN D. COMRIE.

**THE SURGERY OF BASEDOW'S DISEASE.** (*Zur Chirurgie der (403) Morbus Basedow.*) KURT SCHULTZE, *Mittheil. aus den Grenzgeb. der Medicin und Chirurgie*, Juli 1906.

THIS paper takes the form of a report on fifty cases on which the writer had operated, the condition prior to and after operation being fully noted in each case. From his experience of the disease, he is a strong advocate of surgical treatment. Of the 50 cases, 36 were permanently cured, *i.e.* 72 per cent. exhibited no objective or subjective signs of the disease after periods varying from 1-18 years, and expressed themselves as entirely



cured; 12 per cent. were much improved; 2 per cent. remained in *statu quo*; while 12 per cent. died. The operation was the same in all—thyroidectomy; in no case was ligature of the vessels or resection of the sympathetic tried.

It is interesting, further, to note that from a table in which the disease is classified according to the severity, viz. severe, medium, and mild, the earlier surgical treatment is carried out the better are the results. Thus, in the first group, the percentage of recoveries was 57, compared to 100 in the third group and 66 in the second. The mild cases are first treated by medical measures, i.e. medical means are given a trial, and continued with until it is obvious that the disease is not improving, and that prognosis as to operation is getting worse. Of the medical measures he advocates, only two are of real service, viz. Anthyroidein (Möbius), i.e. blood serum of thyroidectomised sheep, and Rodagen, i.e. milk of thyroidectomised goats. The former was used in about 60 cases, and, generally speaking, the results of its administration were good. The subjective symptoms were mainly improved, i.e. palpitations, restlessness, sleeplessness, and mental anxiety were diminished; but a favourable effect was noticed also in the size of the gland itself, its vascularity decreased, the eyes became less prominent and the pulse-rate fell. This improvement, however, was only temporary, as on the first signs of stopping the drug, all the symptoms returned, and in some cases were more aggravated. Eulenburg reported 7 such cases, and gave his opinion of the drug as follows: Its value is limited; it seems to be only a palliative measure. The results obtained with Rodagen were much the same in character. It is a bulky powder, requires to be administered in large doses, and is not very stable.

Dr Schultze found that the operation was more successfully carried out under local anæsthesia, and rapid improvement soon occurred. Rise of temperature was noticed in some of his cases, but it soon fell again. He considers it caused by bronchitis, set up by the mechanical irritation of the trachea, especially if the tumour is a large one and the trachea displaced. Large vessels are encountered, and have to be ligatured in close contact with the trachea, the mucous membrane of which becomes œdematous, and consequently the ciliæ of the lining epithelium become clogged, and mucus is allowed to collect in the bronchi. After a few days, however, this passes off. The pyrexia may also be attributed to the absorption of clots, antiseptics in the wound, and possibly to a fresh output of the existing secretion in the gland. Tetany and cachexia were never observed. The cause of death invariably was due to cardiac failure, and the adoption of local anæsthesia diminished the percentage mortality.

Turning to a consideration of the individual symptoms, we find that the most constant sign recorded was the enlargement of the thyroid gland. This was noticed in 100 per cent. of his cases. It was generally very marked, in some cases being as large as a foetal head. It was further very vascular, and in many cases a thrill could be detected by the hand. In half his cases it interfered with respiration.

The second cardinal symptom, exophthalmus, was found in 92 per cent.; it always followed the appearance of the enlarged thyroid, and only occasionally was it the first sign to be observed. As a rule it was not very marked, but in the more advanced cases caused pain, disturbance of vision, and increased secretion of tears. Both eyes were equally prominent. Graefe's, Stellwag's, and Möbius' signs were noticed in 15, 11, and 7 of the 50 cases. In only 3 cases were they all noticed together.

The most serious cases were those in which the cardiac symptoms were most marked. Tachycardia and palpitations occurred in nearly every case in combination. On three occasions the palpitations existed without tachycardia. In half of the cases no organic change could be found in the heart; in the other half they were only slight, and consisted chiefly of hypertrophy. Oedema and epistaxis were noted five and seven times respectively. These symptoms on the side of the heart show best perhaps the benefits of surgical interference.

Muscular tremors were noticed in 33 cases; they varied in intensity, and only occurred when the patient was spoken to or watched. In practically every case they disappeared after thyroidectomy. Nervous and psychical conditions, *e.g.* mental depression, melancholia, mania, headache, etc., were also favourably influenced.

Eighteen cases exhibited emaciation of varying severity. In a few it constituted a most alarming symptom—one patient lost 14 lb. in twenty-two days. It was generally attributable to diarrhoea and vomiting. In many cases apathy and general debility were well marked.

With regard to the technique of the operation and the extent to which the gland is excised, no mention is made by Dr Schultze.

C. B. PAUL.

---

#### REPORT OF THE CONGRESS OF ALIENISTS AND NEUROLOGISTS OF FRENCH-SPEAKING COUNTRIES

Held at Lille, August 1906.

At the International Medical Congress of 1889 the section of Psychiatry resolved to perpetuate itself in an annual reunion of the alienists of French-speaking countries. Under the auspices of the Société Médico-Psychologique of Paris, was then formed this

annual Congress for the study of the care of the insane, the interests of the personnel of the public establishments in which they were detained, of the relationship of the foregoing to the law, of other aspects of legal medicine, and of the scientific research in mental diseases. A few years later, Neurology was added to the subjects of its consideration, and the anatomo-pathologic aspect became more and more prominent at the meetings; and although the sections separated for the International Congress of 1900, they have reunited once more, and really form a firm body containing most of the leading spirits of France who are engaged in the study of the diseases of the nervous system.

As pointed out by Professor Grasset of Montpellier in his presidential address, it is the neurologists who at present are the chief users of mental measures in the treatment of disease, while the anatomo-pathology of the brain receives fully as much study from the psychiatrists. But indeed the two branches are inseparable.

The gathering has its social functions as well as its purely business one, and the men are most cordial to strangers, among whom was the writer. The chief feature is the three reports.

These consist of subjects decided upon at the previous Congress. One is on Psychiatry, one on Neurology, one on Legal Medicine. The first of these, on the blood in the insane, was made by Dr Maurice Dide of Rennes, where last year's Congress was held. Among the more interesting features he noticed a lowering of the specific gravity immediately before the fit in epilepsy, with a rapid return to the normal, and an increased density in demential conditions. During the epileptic fit the potassium salts are increased, although its alkalinity is decreased. His researches into modification of coagulability are not sufficiently complete for definite results, although he often found coagulation to occur in less than a minute during the stuporose stage of dementia præcox, while in normal subjects he has always found it to occur in from eight to ten minutes.

It is to be regretted that no original data of relative acidity were given, nor of the richness in chlorides. For the latter he merely quoted certain experiments made along with Stenuit in 1898, when the importance of acidoses and of hyperchloruration were not appreciated, while he confined himself to adverse criticism of the technique of Lambranzi, Cappalletti and Lui, and of Charon and Briche.

Biliary toxæmia is the only malady where the changes of the hæmatocytes interest particularly the psychiatrist. The polynuclear cells are increased at the beginning of toxi-infectious psychoses and in states of intoxication. Mononucleosis with inversion of the formula is of great diagnostic importance, being

uncommon in general diseases. It occurs in states of depression and stupor, where the polynuclears may fall to 45 per cent. In epilepsy, Dide thinks that there is a tendency to a diminution of polynuclears during the fit and an increase after it. This is in harmony with the results found in dementia præcox by himself, as well as by Bruce and Peebles, to whose results Dide attached great importance. All these data go to show that both epilepsy and dementia præcox are paroxysmal toxic infections, although Sabrazes pointed out the discordance between the severe symptoms and the slight blood changes, while even these disappear as the malady progresses. He alluded to the causes of error in Ehrlich's tri-acid stain and the hæmoglobinometer of Gowers. He was sorry that the large lymphocytes had not been studied, and thought that Dide's facts do not at all demonstrate that the blood changes have anything to do with the psychosis, especially as it is very probable that the so-called lymphocytes found in tabes, general paralysis, and other chronic diseases are in reality tissue elements, while the toxi-infectious nature of tetany is extremely unlikely in view of its known production by removal of the para-thyroids. To this Dide replies that in 150 autopsies he never found altered para-thyroids, though the thyroids were often diseased.

He then turned to the bacteriology of the blood in the insane, and reported the finding of organisms in a considerable percentage of patients, although the germs are not pathogenic. This statement met with much criticism at the hands of Sabrazes and of Maurice Faure of Paris, who blamed the technique and gave his own results where only eight cases out of a hundred produced microbes, and all were proved to arise through faulty technique, with the exception of one case of typhoid.

Dide replied that Faure's research was on dead bodies, and that he used too small a quantity of bouillon for insemination. The discussion elicited the failure of any observer to find the *spirochaeta pallida* in cases of general paralysis.

As to the serum, Dide never found bile in melancholia, as did Gilbert in the hospitals. In any case the toxicity is experimentally shown to depend on its rapidity of injection as well as on the relative weight of the subject. Its increased toxicity in epilepsy alleged by Carantzine is subject to this critique.

The serum of psychopaths modified the hatching eggs of fowls more than did that of normal individuals (Féré).

The bactericidal power has only rarely been found less. Dide's experiments incline him to accept the doctrine of poly-morphism.

Turning to serology Dide's own experiments showed a notable diminution of alexin in dementia præcox and general paralysis. In not one out of twenty-three cases of the former and four of the latter did complete hæmatolysis occur, although the controls shewed

its invariable completeness. He uses this result to explain his frequent finding of saprophytes in the blood in dementia præcox. He agrees with Bordet in thinking that alexin is of uniform composition, and that specific differences in its action are accounted for by "les sensibilatrices," i.e. the bodies which resist heat up to 65-70°, which act as "specific mordants" in enabling alexin to act.

In the analogy which has been often pointed out between the action of these bodies and that of enterokinase, Dide seeks the explanation of sitiophobic phenomena, an idea he frankly attributes to the reading of Pavlov.

Although in dementia præcox, Dide obtained hæmatolysis with the procedure of Bordet and Gengon, yet his controls shewed results very similar, and he inclines to think that sensibilatrices, like bacteria, are less specific than imagined. He cites the cases where reaction to the tubercle bacillus occurred in people free from all other appreciable sign of tuberculoses. To the writer, this last seems an unhappy example, being given the frequency of latent tubercular lesions.

With regard to neurolynsins and other cytotoxins, Dide merely resumes some of the known work. The very irregular differences of reaction even in the same kind of insanity, he attributes to the variation in the amount of conservation of the undifferentiated reaction of defence common to all organic cells and its capability of stimulation even near the end of life. This reaction diminishes in the following degree; most of all in the acute or subacute confusional psychoses, next in precocious dementia, and least of all in general paralysis. These arguments, if valid, do not support Ford Robertson's explanation of the pathology of this last affection. Among the psychiatric conclusions which Dide draws are the following:—Firstly, that the délire of all the infections and acute intoxications is one, and should for the future be described as one. It is the manifestation of a direct attack upon the thinking cells by toxins conveyed by the blood, and the nature of the reaction depends much more upon the nature of the cell than upon the variety of the toxin.

The sub-acute toxic infectious psychoses are not due to the direct attacks of toxins, but to their indirect influence in diminishing the anti-toxic function of the host. They are not specific. Into the next group general paralysis would fall if Ford Robertson can maintain his position. Its pathogeny is the same, plus the alteration of the supporting tissues. Here the nomenclature of Fournier "parotoxi-infectious" and its connotations is accepted by Dide, who, however, modifies the doctrine in denying specificity to the processes, thus placing in the same group general paralysis and dementia præcox. He thinks this latter is a clinical entity, much more on account

of the clinical signs it presents than from its psychic symptoms, there being other varieties of chronic hallucinatory delire. In this opinion he differs from Deny, who attaches little importance to the physical manifestations. Dide looks upon epilepsy as a cyclical toxæmia, claiming this is proved by the blood changes he has found. The psychoses of involution are for him secondary to hepatic and thyroid alterations as well as to the so-called senility of the brain. But the psychoses of delusional bases are purely psychological in nature. He looks for rational therapeutics of insanity to such agents as will powerfully raise the defensive function of the body cells.

The second report was that on the senile brain, made by Léri, from the Bicêtre laboratory. It was rather hastily put together, but nevertheless embodied results of several years' research by other workers.

Its chief interest lay in the question of the lacunæ and their pathogeny, which the reporter attributes to parenchymatous atrophy in the neighbourhood of small vessels. This conclusion was strongly contested by Anglade, of Bordeaux, who brought preparations to show that the pathogeny of the lacunæ is similar to that of the pulmonary cavity, in being due to the necrosis of an inflammatory area not always in the neighbourhood of a vessel. The process begins by neo-formation and collection of giant multinucleated arthrocytes of which the protoplasm is visible and which possess thick processes formed of groups of fibrils. This stage is followed by disappearance of cells and nuclei and the leaving merely of groups of fibres forming a sclerosis, which in turn begins to crumble, leaving a lacuna in the centre. In the meanwhile, the first stage has been extending at the periphery, and the lacuna enlarges unless cicatricial limitation occurs, as in the young. These processes are most easily seen in the cerebellum. According to Anglade, the worm-eaten appearance so often seen under the ependyma is similarly produced.

Passing to theoretical considerations, Anglade supposed that the nerve cells were in a constant state of struggle with the neuroglia, and that eventually the latter triumphed, if not through disease, at least when old age supervenes. In any case, the preparations he brought strikingly show this interstitial encephalitic reaction. Léri, on the contrary, thought that the apparent crowding of the neuroglia cells was explicable by the lessened volume of the brain, his preparations showing that the corpus callosum, for instance, was diminished fully one half. He denies their neuronophagic function. He distinguishes between toxic and inflammatory proliferations. The diffuse form of sclerosis, all agreed to be non-uniform, and Léri thought it always moderate in amount. The five cases published of miliary sclerosis with fits of senile epilepsy were alluded to. He is of the opinion

that most of these lesions are due rather to the prolonged actions of toxins than to simple senile involution.

In support of this, Hussnot, of Bordeaux, stated that the suprarenals, far from atrophying with the advance of age, were very frequently adenomatous; and he attributes the arterial sclerosis of old age, and cases of premature senility to the increase of the secretion of this gland, which has been proved by experiments in animals to cause arterial sclerosis. The familiar headache, tinnitus, vertigo, amnesia, passing aphasia, slight apoplexy, not followed by hemiplegia, were cited, as well as their tendency to relapse and complication with pseudo-bulbar symptoms. The danger of mistaking arterio-sclerosis for neurasthenia was not emphasised, and *vice versa*, although the rapid fatigability was.

The symptoms of these lesions are usually intermittent, due to the claudication of the cerebral vessels. Those of the motor apparatus, consisting generally of slight paraplegias with "le march aux petits pas," and the dragging or tottering walk of old age are apt to be confounded with those due to similar diffuse sclerosis in the spinal cord, as Raymond pointed out in quoting the researches of Lejonne and Lhermitte. Grasset pointed out that such paraplegics are astonished to find that they can lift the foot and walk like an ordinary individual when they give their attention to doing so specifically. The muscular power, too, of each segment of the limb is perfectly conserved. Anglade, too, pointed out how often the cerebella contained lacunæ, and how this fact explained many senile nervous troubles.

These clinical signs differentiate this form from that due to lesion of the cord and that due to lesions of the cerebral hemisphere proper. In this polygonal form, as Grasset calls it, the lesion is due to multiple lesions in the corpus striatum, as the researches of the Bicêtre laboratory have shown. While Léri has generally found the reflex in extension, Anglade stated that he had always found it in flexion in the slight hemiparaplegia of the aged.

Meige pointed out the therapeutic importance of this distinction, for walking could be retaught to a patient who had lost nothing but his automatic movements.

Anglade tried to indicate the psychoses corresponding to these lesions. For him the senile general paralysis was a clinical reality; it occurred when an intellectual enfeeblement was accompanied by meningeal lesions, the diffuse sclerosis producing a dementia with maniacal, melancholic, or paranoic excitation with an intermittent jargon-aphasia and paraphasia, while in general the lacunar lesions only cause dementia when they affect the cortex.

Regrets were expressed that so little mention was made of the trembling, the troubles of the sensibility, and the modification of the reflexes.

Among the miscellaneous communications, great interest was caused by that of Professor Brissaud on the *rôle* of traumatism in general paralysis. The discussion of this occupied nearly a whole morning, and numerous classic cases were related, as well as more recent personal experiences. It is strange that the question that has been so long decided in Germany should occupy so much valuable time, for only Vallon dissented from the view that traumatism was merely an accident, and had no etiological significance. Indeed, Briand went so far as to ask Brissaud to change the title of his paper into "general paralysis as a factor in the production of traumatism," relating a particularly striking case of an officer who became a general paralytic immediately after a fall from one of the dangerous horses he had latterly been in the habit of riding. At the regimental audit, some weeks afterwards, it was discovered that this officer had been wasting the funds in his care by ordering from Spain a horse of extravagant price, of which the regiment had no need. Brissaud, in all seriousness, refused to change the title of the paper.

Vallon, in a long, rambling speech, conveyed the opinion that traumatism could produce general paralysis. He gave no real facts, and did not even quote, as an analogy, the physiologist's experiment, where, after artificial trauma, one cerebral hemisphere of a dog which had recovered fully from the paralysis thus induced, became once more paralytic on the injection into the blood of bacterial toxins, thus showing the increased susceptibility of traumatised tissues.

In the section of psychiatry, two studies of dementia precox by Mdlle. Pascal, of Ville Evrard, raised questions of clinical importance in the early diagnosis of this much-disputed affection. She thinks that characters of their own are shown by the fits which occur early in dementia precox, and that they are very often mistaken for hysteria, especially as the same patients often at other moments exhibit the silly laugh so often called hysterical.

Professor Regis thought that this laugh is rarely absent early in dementia precox; but he did not agree with Pascal in thinking that a laugh specially excited by solemn events belongs to this psychosis; he thought it was due to the obsession of contrast. He thought, too, that not only precocious dementals presented such convulsions but that they occurred in all the psychoses of intoxication, and were due to toxæmia.

To this Pascal replied that such fits occurred in cases which showed no mental confusion or other toxic symptoms. To those who accept as clinical entities neither dementia precox nor *confusion mentale*, these arguments will not appear of great value. But it must be remembered that only in acute attacks of toxemia does one find such a definite syndrome as the *confusion mentale*, while precocious dementia—a chronic, slowly-progressing malady



—presents psychic signs of intoxication so attenuated that their validity is even denied. But when we reflect that in a disease of definite morbid anatomy so well known as general paralysis, *délire* is neither constant nor always takes the same form, running the gamut between the most exalted grandiose ideas and a depression even leading to suicide, the polymorphism of the early manifestations of dementia precox, and their resemblance to certain phases of confusion mentale, is no reason for separating into numerous categories the admirable synthesis which Kräpelin has given to psychiatry.

Another communication was that in which Williams maintained that traumatic neuroses were merely the products of unskilful suggestion, direct or indirect, by doctors. In support of the contention he quoted a railway accident in America, where out of 200 people (of whom 120 were wounded), only 24 had traumatic neuroses. All those who happened to fall first into the hands of Professor Bevan of Chicago were cured at once, while none of the others were cured until they had received indemnity through the courts. The means of treatment employed by Bevan were, in principle, those of Bernheim, though they were more crudely carried out. Dejerine has used the same in removing what he calls false gastropathies. Their essence is to neutralise the ideas fixed in the patients by the unskilful suggestions of other doctors, either unconsciously created in the course of medical treatment, or acquired by the patient from a clinical picture (in the air, as it were), this of course being primarily of medical painting, "the so-called auto-suggestion."

He was not surprised that these considerations had not become current, in view of the fact that the observations even of an observer like Babinski, about the so-called stigmata of hysteria, had not been better received even by neurologists. He accounts for this by the difficulty the human mind has in freeing itself from such a suppressed major premise as the authority of a text-book. He shows how suggestibility is in proportion to ignorance or laziness of the critical power, and that in medical matters the laity fall an easy prey on that account; and in consequence this type of suggestibility is not a mark of hysteria or neurasthenia. The difficulty of curing these cases is due to the suffering produced by an attempt on the patient's part to modify the false convictions of which he is a victim; for change of convictions means change of personality; and the conservation of the sentiment of the personality is a means towards self-preservation. Cases are quoted in support of this thesis; and the practical application of the paper is to urge the medical profession to counteract the injurious suggestions of charlatans, with which Williams classes those at the bottom of the manifestations of traumatic neuroses. As a first step towards this, he advocates the instruction of the medical

profession in psychology, without which they are quite incapable of explaining to the laity what is in reality not at all mysterious.

Joire, of Lille, showed an apparatus which he called the sthenomètre, by which he claimed to be able to diagnose neurasthenic states through a dynamic influence exerted by the patient on a very light, finely balanced needle in a glass case. He had eliminated as causal factors heat, light, and magnetism, and thinks the deviation is proportional directly to the nervous force of the subject tested. The general verdict of the members was *pas sérieux*. He also read an exceedingly long paper on simulation by hysterics, entering into the whole question from the beginning; but it will be more convenient to discuss this next month, in connection with the third report (of Leroy), on the responsibility of hysterics.

The study of hysteria, which has within recent years fallen into abeyance, after the stimulation it received at the hands of Charcot and his followers, is again beginning to excite attention in France; and, indeed, one of the reports decided upon for next year is on the nature of this psychosis (I use this term advisedly). It will be reported upon by Claude, who has the unsurpassed opportunity of the clinic of Charcot's successor, Professor Raymond, at the Salpêtrière.

This interest is chiefly due to the progress made by the ideas of Babinski, who confines the name hysteria to those manifestations susceptible of production by suggestion, and removable by persuasion. He excludes, therefore, such symptoms as polyuria, cyanotic oedema, and hyperthermia. He claims that misunderstood organic conditions or faulty control of the opportunity for deception by the patient are responsible for the cases reported.

TOM A. WILLIAMS.

(To be continued.)

## Bibliography

### ANATOMY

- VLADISLAV RUZICKA. Berichtendes zur Histologie des zentralen Nervensystems. *Arch. f. mikros. Anat.*, Bd. 68, H. 4, 1906, S. 683.
- SCHIEFFERDECKER. Neurone und Neuronenbahnen. Barth, Leipzig, 1906, M. 11.
- QUENSEL. Beiträge zur Kenntnis der Grosshirnfaserung. *Monatsschr. f. Psychiat. u. Neurol.*, Bd. 20, H. 3, 1906, S. 266.
- LUDWIG UNGER. Untersuchungen über die Morphologie und Faserung des Reptiliengehirns. *Anat. Hefte*, Abt. I., H. 94, 1906, S. 269.
- MÉNCL. Einige Beobachtungen über die Rongoronischen Fibrillen der Nervenzellenkerne. *Arch. f. mikros. Anat.*, Bd. 68, H. 4, 1906, S. 527.
- GEIST. Ueber den "Lobus cerebelli medianus." *Neurol. Centralbl.*, Sept. 16, 1906, p. 855.
- VAN GEHUCHTEN. La Région du lemniscus latéral ou région latérale de l'isthme du rhombencéphale. *Le Névaxe*, Vol. viii., fasc. 1, p. 89.
- LUIGI DE GAETANI. Del nervo intermediario di Wrisberg e della corda dell timpano. *Le Névaxe*, Vol. viii., fasc. 1, p. 67.

- MAX VON FREY. The Distribution of Afferent Nerves in the Skin. *Journ. Amer. Med. Assoc.*, Sept. 1, 1906, p. 625.
- LÉRI. Le cerveau sénile. *Rev. Neurol.*, Aug. 30, 1906, p. 756.
- DOGIEL. Die Endigungen der sensiblen Nerven in den Augenmuskeln und deren Sehnen beim Menschen und den Säugetieren. *Arch. f. mikros. Anat.*, Bd. 68, H. 4, 1906, S. 501.
- J. T. GRADON. Researches on the Origin and Development of the Epiblastic Trabeculae and the Pial Sheath of the Optic Nerve of the Frog, with Illustrations of the Variations met with in other Vertebrates. *Quarterly Journ. of Micros. Sc.*, Aug. 1906, p. 479.
- CARL HUBER. On a Rapid Method of Preparing Large Numbers of Sections. *Ztschr. f. wiss. Mikroskopie*, Bd. 23, H. 2, 1906, S. 187.
- HANS FREUD. Neuer Apparat zur Massenfärbung mikroskopischer Präparate von F. Hellige & Co. *Ztschr. f. wiss. Mikroskopie*, Bd. 23, H. 2, 1906, S. 197.
- WAKELIN BARRETT. The Staining Act: An Investigation into the Nature of Methylene Blue-Eosin Staining. *Bio-Chemical Journ.*, Sept. 1906, p. 406.
- HOPPE. Zur Technik der Weigert'schen Gliafärbung. *Neurol. Centralbl.*, Sept. 16, 1906, p. 854.
- LOBENHOFFER. Ueber die Ergebnisse der Altmann-Schridde'schen Färbemethode beim Zentralnervensystem. *Arch. f. mikros. Anat.*, Bd. 68, H. 4, 1906, S. 491.

### PHYSIOLOGY

- C. K. MILLS. Cerebral Localisation and the Study of Psychiatry. *Brit. Med. Journ.*, Sept. 29, 1906, p. 748.
- RICHARD. Überblick über den heutigen Stand der Frage nach der Lokalisation in der Grosshirnrinde und ihre Anwendung in der forensischen Praxis. *Monatsschr. f. Psychiat. u. Neurol.*, Bd. 20, H. 3, 1906, S. 280.
- PAGANO. Le funzioni del nucleo caudato. Contributo alla psico-fisiologia delle emozioni e all'innervazione centrale degli organi genitali. *Riv. di Pat. nerv. e ment.*, Vol. ii., fasc. 7, 1906, p. 289.
- DRIESCH. Studien zur Entwicklungsphysiologie der Bilateralität. *Arch. f. Entwicklungsmechanik d. Organismen*, Sept. 1906, S. 756.
- JÄDERHÖLM. Untersuchungen über Tonus, Hemmung und Erregbarkeit. *Arch. f. die ges. Physiol.*, Bd. 114, H. 3-4, 1906, S. 248.
- CLUZET. La loi d'excitation des nerfs. *Ann. d'Electrobiol.*, août 1906, p. 505.
- HOORWEG. Ueber die elektrische Erregung der Nerven und der Muskeln. *Arch. f. die ges. Physiol.*, Bd. 114, H. 3-4, 1906, S. 216.
- FÈRE. Le précision du mouvement sous l'influence des excitations. *Compt. Rend. des Stances de la Soc. de Biol.*, T. lx., p. 377.
- TAWARA. Das Reizleitungssystem des Säugetierherzens. Eine anatomisch-histologische Studie über das Atrioventrikulärbündel und die Purkinjeschen Fäden. Fischer, Jena, 1906, M. 10.
- BOTTAZZI. Ein Warmblütermuskelpreparat das sich für Untersuchungen allgemeinen Muskelphysiologie besonders eignet. *Ztschr. f. Biol.*, Bd. 48, H. 3, 1906, S. 432.
- PIERACCINI e CENI. Il tetano faradico e la reazione galvano-muscolare studiate nei rapporti col lavoro muscolare svolgentisi liberamente nel campo del lavoro quotidiano. *Ric. crit. di Clin. Med.*, sett. 15, 1906, p. 581.
- KÜLBS. Experimentelles über Herzmuskel und Arbeit. *Arch. f. Experiment. Pathol.*, Bd. 55, H. 4-5, 1906, S. 288.
- HENRICI. Ueber respiratorische Druckschwankungen in den Nebenhöhlen der Nase. *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. 41, H. 4, 1906, S. 283.
- VON DER VELDEN. Zur Pharmakologie des Nervus Depressor. *Arch. f. Experiment. Pathol.*, Bd. 55, H. 4-5, 1906, S. 223.
- ROUX et HEITZ. De l'influence de la section expérimentale des racines postérieures sur l'état des neurones périphériques. *Nouv. Icon. de la Salpêtrière*, Vol. xix., No. 4, p. 297.

### PSYCHOLOGY

- A. BINET. Cerveau et pensée. *Arch. de Psychol.*, juillet-août 1906, p. 1.
- DECROLY et DEGAND. Les tests de Binet et Simon pour la mesure de l'intelligence. *Arch. de Psychol.*, juillet-août 1906, p. 27.
- CORNELIUS. Psychologische Prinzipienfragen. I. Psychologie und Erkenntnistheorie. *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. 42, H. 6, 1906, S. 401.

- ISADOR H. CORIAT. Experimental Synthesis of Dissociated Memories in Alcoholic Amnesia. *Journ. Abnorm. Psychol.*, Vol. i., No. 3, 1906, p. 109.
- KATZ. Experimentelle Beiträge zur Psychologie des Vergleichs im Gebiete des Zeitsinns. (Schluss.) *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. 42, H. 6, 1906, S. 414.
- LOHMANN. Ueber Helladaptation. *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. 41, H. 4, 1906, S. 290.
- HENRY J. WATT. Ueber die Nachbilder subjektiv gleich heller, aber objektiv verschieden stark beleuchteter Flächen. *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. 41, H. 4, 1906, S. 312.
- AUDIFFRENT. De la Sensation et de l'emotion. *Arch. d'anthropol. crim.*, T. 21, Nos. 151-153, 1906, p. 481.
- PROBST. Les dessins des enfants Kabyles. *Arch. de Psychol.*, juillet-août 1906, p. 131.
- PICK. Sur la confabulation et ses rapports avec la localisation spatiale des souvenirs. *Arch. de Psychol.*, juillet-août 1906, p. 141.
- MAEDER. Contributions à la psycho-pathologie de la vie quotidienne. *Arch. de Psychol.*, juillet-août 1906, p. 148.

## PATHOLOGY

- SOUKHANOFF et PETROFF. Un cas de microcéphalie avec autopsie. *Le Névrose*, Vol. viii., fasc. 1, p. 1.
- GEORG EISATH. Ueber normale und pathologische Histologie der menschlichen Neuroglia. *Monatsschr. f. Psychiat. u. Neurol.*, Bd. 20, H. 3, 1906, S. 240.
- MARINESCO. Quelques recherches sur la morphologie normale et pathologique des cellules des ganglions spinaux et sympathiques de l'homme. *Le Névrose*, Vol. viii., fasc. 1, p. 7.
- SCHAEFFER. Das Verhalten der fibrillo-retikulären Substanz bei Schwellungen der Nervenzellen. *Neurol. Centralbl.*, Sept. 16, 1906, p. 834.
- SAITO. Ueber Dauerverkürzungen an gelähmten Muskeln. *Ztschr. f. Biol.*, Bd. 48, H. 3, 1906, S. 340.
- MÜNZER. Das Waller'sche Gesetz, die Neuronenlehre und die autogene Regeneration der Nervenfasern. *Ztschr. f. Heilk.*, Bd. 27, H. 8, 1906, S. 297.
- EUGENIO RIGNANO. Die centro-epigenetische Hypothese und der Einfluss des Centralnervensystems auf embryonale Entwicklung und Regeneration. *Arch. f. Entwicklungsmechanik*, Sept. 1906, S. 792.
- BABES et MARINESCO. Histologie des lésions expérimentelles et pathologiques des cellules nerveuses surtout des ganglions spinaux. Hirschwald, Berlin, 1906, M. 14.
- HANS EVENSEN. The Pathology of General Paralysis. *Rev. Neurol. and Psychiat.*, Sept. 1906, p. 616.
- HUGO STALBERG. Pathologisch-anatomische Veränderungen des Gehirns bei Lepra, Leprabacillen in Gasser'schen Ganglien und in die Anatomie und Pathologie der Nervenzellen des Gehirns im Allgemeinen. *Arch. f. Psychiat. u. Nervenkt.*, B. 41, H. 3, 1906, S. 809.
- BABONNEIX. Les kystes hydatiques du cerveau chez l'enfant. *Rev. mens. des mal. de l'enfance*, sept. 1906, p. 385.
- BREGMAN. Beiträge zur Pathologie der Varol'schen Brücke. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 31, H. 1-2, 1906, S. 86.
- RUPPEL. Ueber den *Diplococcus intracellularis meningitidis* und seine Beziehungen zu den Gonococcen. *Deutsche med. Wchnschr.*, Aug. 23, 1906, S. 1366.

## CLINICAL NEUROLOGY AND PSYCHIATRY

## GENERAL—

- HERM. MEYER. Compendium der Neurologie und Psychiatrie. Speyer & Kaerner, Freiburg, 1906, M. 3.
- EICHHORST. Pathologie und Therapie der Nervenkrankheiten. 1. Hälfte. Urban & Schwarzenberg, Vienna, 1906, M. 9.
- FÜRNROHR. Die Röntgenstrahlen im Dienste der Neurologie. Karger, Berlin, 1906, M. 10.
- A. T. SCHOFIELD. Mind in Medicine. *Brit. Med. Journ.*, Sept. 29, 1906, p. 765.
- The Nose and the Brain. Leading Article. *Brit. Med. Journ.*, Sept. 29, 1906, p. 792.
- MARTIN BARR. Physical Training a Factor in Psychological Development. *N.Y. Med. Journ.*, Sept. 15, 1906, p. 531.

ASCHAFFENBURG. Die Beziehungen des sexuellen Lebens zur Entstehung von Nerven- und Geisteskrankheiten. *Münch. med. Wchschr.*, Sept. 11, 1906, S. 1793.

T. S. CLOUSTON. The Hygiene of Mind. Methuen & Co., London, 1906, 7s. 6d.

#### MUSCLES—

DREYER. Ueber Skelettveränderungen und Frühkontrakturen bei Dystrophia musculorum progressiva. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 31, H. 1-2, 1906, S. 147.

VON BECHTEREW. Ueber myopathische Muskelhypertrophie. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 31, H. 1-2, 1906, S. 164.

ROSENBERG. Ueber Myatonia congenita. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 31, H. 1-2, 1906, S. 130.

#### PERIPHERAL NERVES—

CHANOZ. Paralyse radiale par fracture de l'humérus. Recherche électrique sur le nerf mis à nu. Guérison malgré un pronostic défavorable. *Arch. d'Électr. Méd.*, sept. 10, 1906, p. 659.

HAUCH. Neuritis puerperalis lumbalis peracuta. *Bibl. f. Laeger*, 1906, p. 13.

JABOULAY. Neurofibromatose avec névrome plexiforme. *Gaz. des Hôp.*, août 28, 1906, p. 1155.

RONNEAUX. Deux cas de zona traités et guéris par l'effluvation de haute fréquence. *Ann. d'Électrobiol. et de Radiol.*, July 1906, p. 493.

OSTERROHT. Herpes zoster ophthalmicus. Marhold, Halle, 1906, M. —80.

#### SPINAL CORD—

JEAN LÉPINE. Rapports du rhumatisme chronique avec quelques maladies de la moelle. *Lyon Méd.*, sept. 16, 1906, p. 465.

**Tabes.**—DUBOIS. Sur la coexistence des accidents syphilitiques tertiaires avec le tabès. *Thèse*. Storck et Cie, Lyon, 1906.

MARTIN. The Sphincter Reflexes in Tabes Dorsalis and Paresis. *Journ. of Nerv. and Ment. Dis.*, Aug. 1906, p. 527.

**Progressive Muscular Atrophy.**—STIEFLER. Zur Klinik der neuralen Form der progressiven Muskelatrophie. *Ztschr. f. Heilk.*, Bd. 27, H. 8, 1906, S. 219.

**Amyotrophic Lateral Sclerosis.**—PUSCARIN et LAMBRIOR. Sclérose latérale amyotrophique avec phénomènes spasmodiques très accentués et amyotrophie accusée des membres supérieurs. Phénomènes bulbaires très marqués, marche rapide, mort. Autopsie. *Rev. Neurol.*, sept. 15, 1906, p. 789.

**Little's Disease.**—GLASCOCK. Report of a Case of Little's Disease. *N.Y. Med. Journ.*, Sept. 1, 1906, p. 433.

**Myelitis.**—SALLE. Zur Frage über die Wege der aufsteigenden Myelitis. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 31, H. 1-2, 1906, S. 108.

**Encephalomyelitis.**—OTTO MARBURG. Die sogenannte akute Sklerose (Encephalomyelitis periaxialis scleroticans). Deuticke, Wien, 1906, M. 3.

**Pott's Disease.**—BOSCHI et GRAZIANI. Un cas de paraplégie pottique. *Ra. Neurol.*, Sept. 15, 1906, p. 799.

**Disseminated Sclerosis.**—HELLER. Zur Differentialdiagnose zwischen psychogener Neurose und multipler Sklerose. *Klin. f. psych. u. nerv. Krankh.*, Bd. 1, H. 3, S. 252.

ASHLEY W. MACKINTOSH. On the Frequency with which Certain Signs and Symptoms occur in Cases of Disseminated Sclerosis before the Development of so-called Cardinal Symptoms. *Rev. Neurol. and Psychiat.*, Sept. 1906, p. 601.

CATOLA. Sclérose en plaques et syphilis. *Nouv. Icon. de la Salpêtr.*, Vol. xix, No. 4, p. 337.

**Syringomyelia.**—BURR. A Note on the Temporary Disappearance of the Sensory Symptoms in Syringomyelia. *Journ. of Nerv. and Ment. Dis.*, Aug. 1906, p. 525.

**Tumour.**—GEORG STERTZ. Klinische und anatomische Beiträge zur Kasuistik der Rückenmarks- und Wirbeltumoren. *Monatsschr. f. Psychiat. u. Neurol.*, Bd. 20, H. 2, 1906, S. 195.

**Concussion.**—CORNER. Concussion of the Spine, with some Remarks on Concussion in general. *Lancet*, Sept. 22, 1906, p. 784.

**Spine.**—PONCET et LÉRICHE. Pathogénie des ankyloses spontanées et particulièrement des ankyloses vertébrales. Rey et Cie, Lyon, 1906.

LERI. Pathogénie des ankyloses et particulièrement des ankyloses vertébrales. Rey et Cie, Lyon, 1906.

## BRAIN—

- Meningitis.**—WESTENHOEFFER. Ueber den gegenwärtigen Stand unserer Kenntnisse von der übertragbaren Genickstarre. *Berl. klin. Wchnschr.*, Sept. 24, 1906, S. 1267.
- BLAU. Kasuistischer Beitrag zur Meningoencephalitis serosa. *Ztschr. f. Ohrenheilk.*, Bd. 52, H. 1-2, 1906, S. 129.
- WESTENHOEFFER. Pathologisch-anatomische Ergebnisse der oberschlesischen Genickstarreepidemie von 1905. Fischer, Jena, 1906, M. 7.
- WRIGHT. The Rash in Cerebro-spinal Meningitis. *Lancet*, Sept. 15, 1906, p. 717.
- Hæmorrhage.**—MARIE et MOUTIER. Deux cas d'hémorragie protubérantielle. *Nouv. Icon. de la Salpêtr.*, Vol. xix., No. 4, p. 383.
- Encephalitis.**—LAACHE. Til encefalit-spøgsmaalet. *Norsk. Magaz. f. Lægevid.*, 1906, p. 1.
- Sinus Thrombosis.**—FRIED. STOCKER. A rare case of Thrombosis of the Cavernous Sinus following Orbital Thrombo-phlebitis. *Arch. of Ophthalmol.*, Vol. xxxv., No. 4, p. 373.
- YERSIN. Phlébite infectieuse du sinus caverneux. *Rev. méd. de la Suisse Romande*, août 20, 1906, p. 456.
- ST CLAIR THOMSON. Cerebral and Ophthalmic Complications in Sphenoidal Sinusitis. *Brit. Med. Journ.*, Sept. 29, 1906, p. 768.
- Tumour.**—OGDEN and MATHEWS. A Case of Sarcoma with Secondary Intracranial Growths in a Child of Five. *Brit. Journ. Children's Dis.*, Sept. 1906, p. 394.
- SALTYKOW. Heilungsvorgänge an Erweichungen, Lichtungsbezirken und Cysten des Gehirns. *Arch. f. Psychiat. u. Nervenk.*, Bd. 41, H. 3, 1906, S. 1053.
- Abscess.**—PAUSE. Ein Fall von Kleinhirnsabscess. *Monatsschr. f. Ohrenheilk.*, Bd. 40, H. 8, 1906, S. 539.
- THÉVENET et ROUBIER. Abscess extra-durémériens. *Gaz. des Hôp.*, sept. 22, 1906, p. 1287.
- CIAMPOLINI. Sintomatologie e diagnosi degli ascessi cerebellari d'origine otitica. *Riv. crit. di Clin. Med.*, sett. 22, 1906, p. 605.
- ALFRED WIENER. A Case of Brain Abscess following Traumatism and Acute Mastoiditis. A Case of Hysteria simulating Brain Abscess after Operation for Secondary Mastoiditis. *Arch. of Otol.*, Vol. xxxv., No. 4, 1906, p. 340.
- ZEBROWSKI. Zur Kasuistik der otitischen Hirnabscesse. *Monatsschr. f. Ohrenheilk.*, Bd. 40, H. 8, 1906, S. 544.
- HÜTTIG. Zur Kasuistik der endokraniellen Komplikationen der Mittelohreiterungen. *Arch. f. Ohrenheilk.*, Bd. 68, H. 3-4, 1906, S. 233.
- General Paralysis.**—SALMON. Della diagnosi differenziale tra la paralisi progressiva e la sifilide cerebrale. *La Clin. Moderne*, agosto 22, 1906.
- RODIET, DUBOIS et PANSIER. Les symptômes oculaires de la paralysie générale. *Arch. de Neurol.*, août 1906, p. 90.
- W. JULIUS MICKLE, ALDREN TURNER, and others. A Discussion on General Paralysis. *Brit. Med. Journ.*, Sept. 29, 1906, p. 741.
- DUCHAMP. Les Troubles oculaires dans la paralysie générale au début. Dirion, Toulouse, 1906, 3 fr.
- M. REICHARDT. Ueber Knochenveränderungen bei progressiver Paralyse. *Centralbl. f. Nerv. u. Psychiat.*, Sept. 15, 1906, p. 705.

## MENTAL DISEASES—

- DIEFENDORF. Etiology of Dementia Paralytica. *Brit. Med. Journ.*, Sept. 29, 1906, p. 744.
- DIDE. Étude cytologique, bactériologique et expérimentale du sang chez les aliénés. *Rev. Neurol.*, aug. 30, 1906, p. 750.
- T. D. CROTHERS. The Insanity of Inebriety. *Brit. Med. Journ.*, Sept. 29, 1906, p. 753.
- SERGE SOUKHANOFF. On Hypochondriacal Melancholia in Russian Soldiers. *Journ. Abnorm. Psychol.*, Vol. i., No. 3, 1906, p. 135.
- C. K. CLARKE, ADOLF MEYER, and F. X. DERCUM. A Discussion on Dementia Præcox. *Brit. Med. Journ.*, Sept. 29, 1906, p. 755.
- CLARENCE B. FARRER. Types of the Devolutional Psychoses. *Brit. Med. Journ.*, Sept. 29, 1906, p. 760.
- MARCHANT et OLIVIER. Diabète et troubles mentaux. *Gaz. des Hôp.*, sept. 6, 1906, p. 1203.
- COURTNEY. On the Clinical Differentiation of the Various Forms of Ambulatory Automatism. *Journ. Abnorm. Psychol.*, Vol. i., No. 3, 1906, p. 123.

- SALGÓ. Die forensische Bedeutung der sexuellen Perversität. Marhold, Halle, 1906, M. 1.20.
- STELZNER. 200 Selbstmordfällen nebst Beitrag zur Prognostik der mit Selbstmordgedanken verknüpften Psychosen. Karger, Berlin, 1906, M. 4.
- FRANCO DE ROCHA. La psychose maniaque dépressive. *Ann. méd.-psychol.*, sept.-oct. 1906, p. 250.
- EVENSEN. Fra de 10 første aar af Kriminalasylets virksomhed. *Tidsskr. f. den Norske Lægeforening*, 1906, p. 61.
- MARCUS WYLER. Beiträge zu einem Grundriss des vergleichenden Irrechten. Marhold, Halle, 1906, M. 2.
- ROBERT RENTOUL. Proposed Sterilization of Certain Mental Degenerates. *Brit. Med. Journ.*, Sept. 29, 1906, p. 765.
- ADAM. Des établissements d'aliénés, d'idiots et d'épileptiques. Du rôle du médecin dans ces établissements. *Ann. méd.-psychol.*, sept.-oct. 1906, p. 263.
- LAQUER. Die ärztliche und erziehlische Behandlung von Schwachsinnigen (Debilen und Imbezillen) in Schulen und Anstalten und ihre weitere Versorgung. *Klin. f. psych. u. nerv. Krankh.*, Bd. 1, H. 3, S. 208.
- VERNON BRIGGS. Observation Hospitals or Wards for Early Cases of Mental Disturbances. *Boston Med. and Surg. Journ.*, Sept. 1906, p. 252.
- WERNER. Die Versorgung der geisteskranken Verbrecher in Dalldorf. Fischer, Berlin, 1906, M. 4.
- HOPPE. Ein gang durch eine moderne Irrenanstalt. Marhold, Halle, 1906, pp. 75, price M. 1.60.

#### GENERAL AND FUNCTIONAL DISEASES—

- Chorea.**—VETLESEN. Chorea gravidarum and psychosis. *Norsk. Magaz. f. Lægevid.*, p. 67.
- MACEY. A Case of Osteitis Deformans with Huntingdon's Chorea. *Lancet*, Sept. 22, 1906, p. 787.
- Epilepsy.**—THALWITZER. Epileptiker als Autofahrer. *Münch. med. Wchnschr.*, Sept. 11, 1906, S. 1818.
- GEORG LOMER. Witterungseinflüsse bei Epileptischen. *Arch. f. Psychiat. u. Nervenk.*, Bd. 41, H. 3, 1906, S. 1009.
- MOON. Some Observations on Convulsions in Children and their Relation to Epilepsy. *Lancet*, Sept. 15, p. 721.
- JULES VOISIN et ROGER VOISIN. Emploi du bromure de potassium dans l'épilepsie. *Presse méd.*, août 25, 1906, p. 541.
- Neurasthenia.**—DUNIN. Ueber den Begriff der Neurasthenie. *Berl. Klin. Wchnschr.*, Sept. 24, 1906, S. 1221.
- RANSCHKE. Die diagnostische Bedeutung hypochondrischer Vorstellung. *Berl. klin. Wchnschr.*, Sept. 10, 1906, S. 1221.
- ROMBERG. Bemerkungen über Neurasthenie und ihre klimatische und balneotherapeutische Behandlung. *Deutsche med. Wchnschr.*, Sept. 20, 1906, S. 1523.
- TERRIEN. L'Hystérie et la Neurasthénie chez le paysan. Siraudeau, Angers, 1906.
- LEMOINE. Traitement de certains cas de neurasthénie par le fer. *Progrès méd.*, sept. 8, 1906, p. 563.
- Hysteria.**—LEROY. La responsabilité des hystériques. *Rev. Neurol.*, Aug. 30, 1906, p. 765.
- BAUMANN. Ein seltener Fall von hysterischem Dämmerzustande. *Neurol. Centralbl.*, Sept. 16, 1906, p. 849.
- TÖRNE. Hysterisk afoni med ensidig recurrensförlamning. *Hygiea*, 1906, p. 258.
- Tetany.**—LEVI. Sopra alcuni nuovi casi de Tetania degli adulti. *Riv. Crit. di Clin. Med.*, agosto 25 e sett. 1, 1906.
- GUINON. Tétanie à forme de tétanos au début de la fièvre typhoïde. *Re. mens. des mal. de l'enfance*, sept. 1906, p. 409.
- STOELTZNER. Kinder-Tetanie und Epithelkörperchen. *Jahrb. f. Kinderheilk.*, Sept. 1906, S. 482.
- MENDELSON und KUHN. Beobachtungen über kühlmilchfrei Ernährung bei dem Laryngospasmus der Tetanie und Eklampsie der Kinder. *Arch. f. Kinderheilk.*, Bd. 44, H. 1-3, 1906, S. 86.
- Neuralgia.**—LEO WILLIAMS. Neuralgia and Headache. *Clin. Journ.*, Aug. 22, 1906, p. 295.
- LEWIS S. SOMERS. Nasal Disease and Neuralgia. *Journ. Amer. Med. Assoc.*, Sept. 8, 1906, p. 741.
- Paralysis Agitans.**—KINICHI NAKA. Zur pathologischen Anatomie der Paralysis agitans. *Arch. f. Psychiat. u. Nervenk.*, Bd. 41, H. 3, 1906, S. 787.

- Vasomotor Disorders.**—ROSENFELD. Zur Kasuistik der vasomotorisch-trophischen Neurose. *Centralbl. f. Nervenheilk. u. Psych.*, Sept. 1, 1906, p. 665.
- Acromegaly.**—GAUSSEL. Un cas d'acromégalie. *Nouv. Icon. de la Salpêtr.*, Vol. xix., No. 4, p. 391.
- HUDOVERNIG. Un cas de gigantisme précoce. *Nouv. Icon. de la Salpêtr.*, Vol. xix., No. 4, p. 398.
- Exophthalmic Goitre.**—GIFFORD. Ueber ein neues Augensymptom bei Morbus Basedowii. *Klin. Monatsbl. f. Augenheilk.*, Sept. 1906, S. 201.

#### SPECIAL SENSES AND CRANIAL NERVES—

- NAGEL. Fortgesetzte Untersuchungen zur Symptomatologie und Diagnostik der angeborenen Störungen des Farbensinns. *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. 41, H. 4, 1906, S. 239.
- CAVAZZANI. Lesioni spinali e riflessi pupillari. *Riv. crit. di Clin. Med.*, sett. 8, 1906, p. 565.
- HÜBNER. Untersuchungen über die Erweiterung der Pupillen und psychische und sensible Reize nebst einigen allgemeinen Bemerkungen über Pupillenreactionen. *Arch. de Psychiat. u. Nerven.*, Bd. 41, H. 3, 1906, S. 1016.
- BAUROWICZ. Eine otogene Abduzenslähmung. *Monatsschr. f. Ohrenheilk.*, Bd. 40, H. 8, 1906, S. 535.
- MÜHSAM. Augenmuskellähmung nach Rückenmarksanästhesie. *Deutsche med. Wchnschr.*, Aug. 30, 1906, S. 1411.
- WENDELL REBER. A Study of Convergence and its Defects, including an Analysis of 441 Cases of Exophoria. *Journ. Amer. Med. Assoc.*, Sept. 1, 1906, p. 670.
- WEYL. Ueber Nystagmus toxicus. *Berl. klin. Wchnschr.*, Sept. 17, 1906, S. 1244.
- KUBO. Ueber die von N. acusticus ausgelösten Augenbewegungen (besonders bei thermischen Reizungen). *Arch. f. die gesam. Physiol.*, Bd. 114, H. 3-4, 1906, S. 143.
- SOHIER BRYANT. The Great Psychical Importance of Ear Disease. *Journ. Nerv. and Ment. Dis.*, Sept. 1906, p. 553.
- EMIL AMBERG. Ear Affections and Mental Disturbances. *Journ. Nerv. and Ment. Dis.*, Sept. 1906, p. 566.
- HOLGER MYGIND. Die Paralyse des Musc. crico-thyroideus. *Arch. f. Laryngol.*, Bd. 18, H. 3, 1906, S. 403.
- GROSSMAN. Beitrag zur Lehre von der wechselseitigen funktionellen Beziehung der Kehlkopfmuskeln untereinander. *Arch. f. Laryngol.*, Bd. 18, H. 3, 1906, S. 463.
- GRABOWER. Zur Recurrensfrage. *Arch. f. Laryngol.*, Bd. 18, H. 3, 1906, S. 419.
- GEORG AVELLIS. Neue Fragestellung zur Symptomatologie der Sensibilitätsstörungen im Larynx. *Arch. f. Laryngol.*, Bd. 18, H. 3, 1906, S. 472.
- ROLLESTON. Precocious Paralysis of the Palate in Diphtheria. *Rev. Neurol. and Psychiat.*, Sept. 1906, p. 608.
- PERSON. Sur un cas d'hémi-paralyse de la langue chez le nouveau-né. *Thèse. Impr. centrale, Soissons*, 1906.

#### MISCELLANEOUS SYMPTOMS—

- STRASBURGER. Zur Klinik der Bauchmuskellähmungen, auf Grund eines Falles von isolierter partieller Lähmung, nach Poliomyelitis anterior acuta. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 31, H. 1-2, 1906, S. 52.
- CURSCHMANN. Beiträge zur Physiologie und Pathologie der kontralateralen Mitbewegungen. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 31, H. 1-2, 1906, S. 1.
- HRŮSKA. Ein Fall von Typhus abdominalis mit nachfolgender Lähmung und Aphasie. *Prag. med. Wchnschr.*, Sept. 20, 1906, S. 487.
- LANG. Lähmungen nach Lumbalanästhesie mit Novocain und Stovain. *Deutsche med. Wchnschr.*, Aug. 20, 1906, S. 1412.
- DAIREAUX. Étude sur les paralyses pneumoniques chez les adultes. *Arch. gén. de méd.*, Sept. 4, 1906, p. 2241.
- HERZOG. Ueber das Vibrationsgefühl. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 31, H. 1-2, 1906, S. 96.
- CHARLES L. DANA. The Question of Protopathic and Epicritic Sensibility and the Distribution of the Trigeminal Nerve (Third Branch). *Journ. Nerv. and Ment. Dis.*, Sept. 1906, p. 577.
- LESEM. Acroparesthesia. A Study of 200 Cases. *Med. Rec.*, Sept. 1, 1906, p. 337.



- PILTZ. Ein Beitrag zum Studium der Dissociation der Temperatur- und Schmerzempfindung bei Verletzungen und Erkrankungen des Rückenmarkes. *Arch. f. Psychiat. u. Nervenk.*, Bd. 41, H. 3, 1906, S. 951.
- ERNEST DORE. On Cutaneous Affections in Various Diseases, with especial Reference to Certain Angio-neuroses. *Brit. Journ. of Dermatol.*, Sept. 1906, p. 305.
- BONNE. Hémigénésie cérébelleuse; agénésie partielle du corps calleux et du lobe limbique; anomalies des circonvolutions cérébrales. *Arch. de Neurol.*, août 1906, p. 65.
- TROMNER. Ein Fall von "Gehstottern." *Neurol. Centralbl.*, Sept. 16, 1906, p. 857.
- ACHILLES ROSE. Is Nervous Dyspepsia a Disease sui generis? *N. F. Med. Journ.*, Sept. 1, 1906, p. 441.
- SCHMOLL. Zwei Fälle von Adams-Stokes'scher Krankheit mit Dissociation von Vorhof- und Kammerrhythmus und Läsion des His'schen Bündels. *D. Arch. f. klin. Med.*, Bd. 87, H. 5-6, 1906, S. 554.
- PARISOT et ETIENNE. Arthropathies séniles des doigts. *Nouv. Icon. de la Salpêtr.*, Vol. xix., No. 4, p. 387.
- NORMAN BRIDGE. Some Truths about Sleep. *Journ. Amer. Med. Assoc.*, Sept. 1, 1906, p. 652.
- VICTOR HAMMERSCHLAG. On Disturbances of Speech in Childhood. *Arch. of Otol.*, Vol. xxxv., No. 4, 1906, p. 379.
- KURT GOLDSTEIN. Zur Frage der amnestischen Aphasie und ihrer Abgrenzung gegenüber der transcorticalen und glossopsychischen Aphasie. *Arch. f. Psychiat. u. Nervenk.*, Bd. 41, H. 3, 1906, S. 911.
- INGENIEROS. Les aphasies musicales. *Nouv. Icon. de la Salpêtr.*, Vol. xix., No. 4, p. 362.
- WORTHINGTON. Some Observations on Stammering and its Treatment. *Lancet*, Sept. 22, 1906, p. 789.
- GÜTZMANN. Das Stottern und seine gründliche Beseitigung durch ein methodisch geordnetes und praktisch erprobtes Verfahren. Staude, Berlin, 1906, M. 2, 50.
- CLAPAREDE. Agnosie et asymbolie à propos d'un soi-disant cas d'aphasie tactile. *Rev. Neurol.*, Sept. 15, 1906, p. 803.

#### TREATMENT\*—

- PAUL DUBOIS. Rational Psycho-Therapeutics. *Brit. Med. Journ.*, Sept. 29, 1906, p. 767.
- PAUL EMILE LEVY. Pathogénie psychique et psychothérapie. Les psychonévroses méconnues. *Journ. des prat.*, août 11, 1906, p. 501.
- BUSCHMANN. Die Massage als Vorbeugungs- und Heilmittel gegen Nervenleiden. Gewerbe-Buchh., Dresden, 1906, M. 1.
- STEYERHAL. Die Ernährung Nervenkranken. *Ztschr. f. Krankenpflege*, Aug. 1906, S. 298.
- GEIJERSTAM. Nagra fall af motoriska neuroser, behandlade med hypnotism. *Hygiea*, 1906, p. 126.
- J. R. O'BRIEN. The Value of Hypnotism as a Therapeutic Agent. *Med. Mag.*, Sept. 1906, p. 453.
- C. S. BULL. The Treatment of Progressive Atrophy of the Optic Nerve due to Acquired Syphilis by Subconjunctival and Intravaginal Injections of Sublimate of Mercury. *Journ. Amer. Med. Assoc.*, Sept. 15, 1906, p. 823.
- ZBINDEN. Influence de l'autosuggestion sur le mal de mer. *Arch. de Psychol.*, juillet-août 1906, p. 153.
- BERNHARDT. Ueber Nervenpfropfung bei peripherischen Facialislähmung vorwiegend vom neurologischen Standpunkte. *Mitt. a. d. Grenzgebiete der Med. u. Chir.*, Bd. 16, H. 3, 1906, S. 476.
- PERS. Om kirurgisk Behandlung af Ischias. *Hosp.-Tid.*, 1906, p. 93.
- BREGMAN. Ein Beitrag zur Klinik und zur operativen Behandlung der Rückenmarksgeschwülste. *Deutsche Ztschr. f. Nervenheilk.*, Bd. 31, H. 1-2, 1906, S. 68.
- SPILLER and FRAZIER. Cerebral Decompression. *Journ. Amer. Med. Assoc.*, Sept. 1, 8, and 15, 1906.
- KUCHNER. Apparat zu Operationsübungen am Schläfenbeine. *Ztschr. f. Ohrenheilk.*, Bd. 52, H. 1-2, 1906, S. 90.
- CARL BECK. On the Use of the Temporal Fascia to Cover in Cranial Defects. *Ann of Surg.*, Part clxiv., 1906, p. 170.

\* A number of references to papers on Treatment are included in the Bibliography under the individual Diseases.

# **Review** of **Neurology and Psychiatry**

---

## **Original Article**

### **A CASE OF PARTIAL DOUBLING OF THE SPINAL CORD.<sup>1</sup>**

By PURVES STEWART, M.A., M.D. Edin., F.R.C.P.,  
Physician in charge of Out-Patients at the Westminster Hospital ;  
Physician to the Royal National Orthopædic Hospital ;  
and

JULIUS BERNSTEIN, M.B. Lond., M.R.C.S.,  
Assistant Pathologist and Curator of the Museum to the  
Westminster Hospital.

THE following case of partial doubling of the spinal cord came under our observation at Westminster Hospital during the summer of 1904.

#### **CLINICAL NOTES.**

The patient, a lad of sixteen, a clerk by occupation, was admitted to the wards under Dr Purves Stewart, suffering from tuberculous meningitis. The family history was unimportant. There was no consanguinity of the parents. The patient had always been observed to walk a little clumsily, but his general health had been good and his intelligence quite up to normal.

Three weeks before admission he began to complain of frontal headache, and the bowels were confined ; two days before admission he began to vomit, and on the day before he came in he became drowsy and developed incontinence of urine.

When admitted on 22nd August he was semi-comatose, but

<sup>1</sup> The pathological part of this paper has already been communicated by one of us (J. B.) to the *Transactions of the Pathological Society of London*, vol. lvii., to which Society we are indebted for the use of Figure facing p. 732.

could be roused with difficulty. The optic discs were normal. The pupils were equal and did not react to light. The other cranial nerves were normal. He had no paralysis of any limb. There was well-marked pes cavus on both sides. The knee-jerks and ankle-jerks could not be elicited. The plantar reflexes were of a doubtful extensor type. The temperature was 100° F., and the pulse 74. The urine was normal.

On 23rd August, lumbar puncture was performed. The intrathecal pressure was found to be markedly increased. The fluid was clear, but on microscopic examination showed a large excess of degenerating lymphocytes.

*Aug. 24.* To-day (apparently as a result of relief of intracranial pressure by the lumbar puncture) the patient's coma has passed off and he is quite rational. The left pupil is slightly larger than the right, and both of them react sluggishly to light. There is occasional transient inward strabismus of the left eye. The face, palate, tongue, upper and lower limbs are normal and symmetrical in their movements. The knee-jerks and ankle-jerks are still absent.

*Aug. 25.* The patient is again semi-comatose. The pupils react very sluggishly to light. The ankle-jerks are just present; the knee-jerks are not elicited. The right plantar reflex is of extensor type, the left flexor.

*Aug. 26.* The coma is now complete. No additional paralysis has developed. There is still occasional transient inward strabismus of the left eye. The spinal muscles also occasionally become tonically contracted. There is no head-retraction. Lumbar puncture withdraws 22 cubic centimetres of clear fluid under excessive pressure.

*Aug. 27.* The patient is again partially conscious. The right eye is fixed in the mid-position, the left occasionally rotates inwards alone.

*Aug. 28.* The pupils are now dilated and insensitive to light.

*Aug. 30.* The pupils are widely dilated and insensitive. The veins of the left optic disc are now fuller than normal, but there is no measurable swelling. The right eye is fixed in the mid-position, whilst the left makes occasional inward movements. The patient is semi-comatose and makes restless movements of the face and hands. There have been no fits. The knee-jerks and ankle-jerks are again absent. The plantars are flexor in

type. The temperature, which since admission has varied from 99° F. to 101° F., is now over 102.2° F.; and the pulse, which on admission was 80, is now 144.

*Aug. 31.* Patient died this morning, with a temperature of 105° F.

#### REMARKS.

The diagnosis made during life was that of basal tuberculous meningitis. The history of habitual clumsiness of gait, together with the presence of pes cavus, suggested the possibility of a co-existing Friedreich's ataxy. The absence of the knee-jerks and ankle-jerks on admission might have seemed to support such a view, but, three days after admission, the ankle-jerks were elicited quite distinctly, and we therefore preferred to attribute the depression of the deep reflexes to increased intracranial pressure. The presence of the pes cavus therefore remained unaccounted for.

#### PATHOLOGICAL APPEARANCES.

At the base of the brain there were the usual appearances of tuberculous meningitis. The spinal meninges were normal.

On opening the vertebral canal a marked bulging of the membranes in the lumbar region was observed. The theca and its contents almost filled the vertebral canal. On opening the theca, its abnormal fulness was found to be due, not to excess of fluid, but to a bifurcation of the spinal cord, commencing at the level of the first lumbar segment and becoming almost complete in the third lumbar segment, at which level, on transverse section, the naked-eye appearance was that of two spinal cords, slightly united at their mesial aspects, each possessing grey and white matter, with two anterior cornua and one posterior cornu on each side. Below this level the two cords gradually fused together again, the dividing fissures becoming progressively shallower, and the cord ultimately became continuous with the filum terminale, which was single, though somewhat thicker than usual.

The cord was fixed in Müller's fluid. After being divided into blocks, each segment was cut in paraffin, serial sections being made of the first lumbar segment, in order to study the question of the bifurcation of the central canal and surrounding grey matter.

The appearances at different levels are shown in Figs. 1 to 11, which were drawn to scale by means of the Edinger projection-apparatus, and in the photographs appended.

In the upper part of the first lumbar segment (see Figs. 1 and 2) the appearances are those of a normal cord. Towards the lower part of this segment, whilst the central canal remains single, the posterior or grey commissure becomes more diffuse and bulges backwards towards the posterior columns. Lower still (see Fig. 2) a smaller canal appears, situated in front and to one side of the main central canal.

In the upper part of the second lumbar segment the cord is divided into two almost complete halves (see Fig. 3) by the deepening of the original anterior and posterior median septa. Each half-cord possesses a central canal towards its inner aspect, a complete anterior and posterior cornu, with, at the outer side, anterior and posterior nerve-roots, and mesially a mass of grey matter, the downward continuation of the original grey commissure, spread somewhat profusely around the bisecting processes of pia mater. Lower still, the two central canals diverge from each other towards the centres of their respective half-cords, but never quite reaching the centre on each side. The mesially situated grey matter assumes the appearances of an anterior and a posterior cornu on each side of the mesial furrow, these new cornua becoming separated from the pia mater by a tract of white matter (see Fig. 4). Meanwhile the separation of the two half-cords becomes more complete, and each develops an anterior fissure and a less perfect posterior septum, the two new anterior fissures being inclined towards each other and towards the original anterior median fissure, which is now antero-mesial with relation to each half-cord.

In the third lumbar segment (see Fig. 6) the separation of the two half-cords is at its maximum. Each has an anterior and a posterior septum, a central canal, two anterior cornua and two posterior cornua. The antero-external cornu on each side is better developed than the antero-mesial cornu. The antero-external cornu possesses well-formed nerve-cells, whereas in the antero-mesial cornu the nerve-cells are small in size and scanty in numbers. Moreover, the anterior nerve-roots are derived only from the antero-external cornua. The two half-cords appear as if rotated inwards, each on its own long axis, so that the two

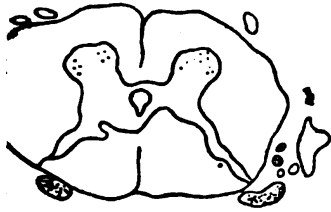


Fig. 1. 1<sup>st</sup> lumbar segment

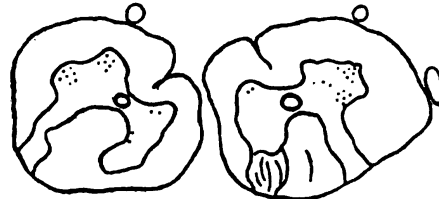


Fig. 6. 3<sup>rd</sup> lumbar segment

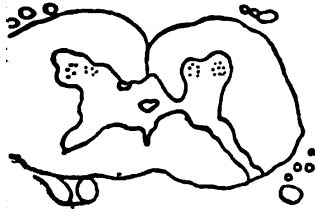


Fig. 2. 1<sup>st</sup> lumbar segment

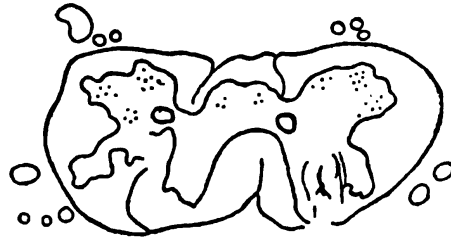


Fig. 7. 4<sup>th</sup> lumbar segment

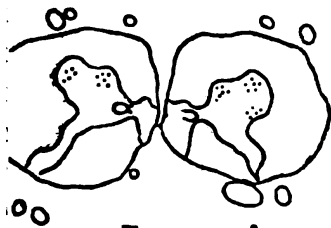


Fig. 3. 2<sup>nd</sup> lumbar segment

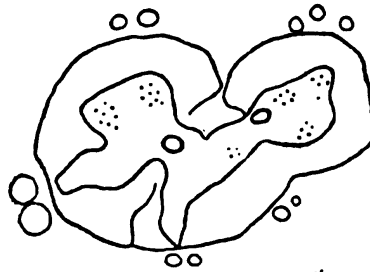


Fig. 8. 5<sup>th</sup> lumbar segment

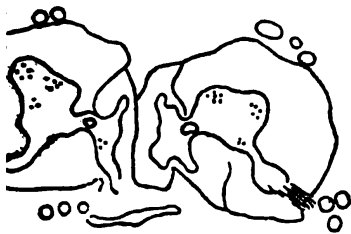


Fig. 4. 2<sup>nd</sup> lumbar segment

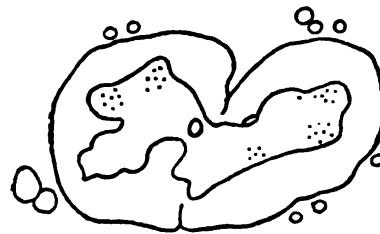


Fig. 9. Upper sacral segment

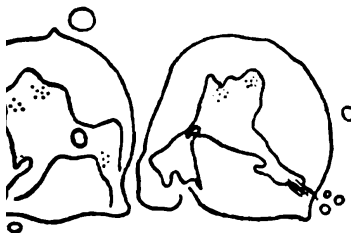


Fig. 5. 2<sup>nd</sup> lumbar segment



Fig. 11. Cervical segment

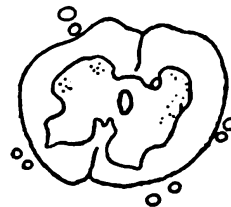


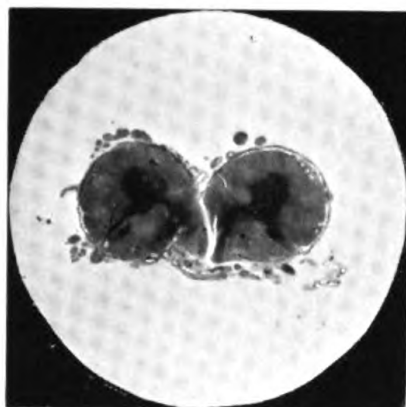
Fig. 10. Lower sacral segment

antero-mesial fissures incline inwards towards each other. The grey matter of the postero-mesial cornua is somewhat diffuse and broken up by irregular strands of white matter.

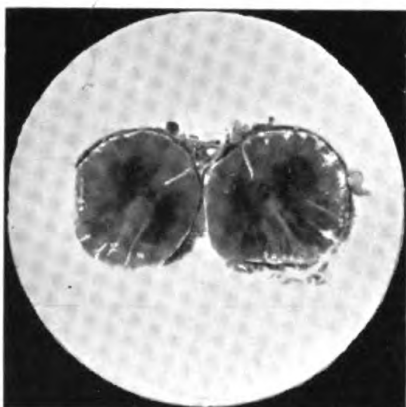
Below this level the two half-cords gradually fuse together again, but in a less symmetrical manner than that in which they had separated. Firstly, the adjacent antero-mesial cornua become fused (see Fig. 7), the two postero-mesial cornua being still separated by white matter, in which is a posterior septum. There are still two central canals and two antero-mesial septa symmetrically situated. Lower down, the two antero-mesial fissures meet to form a single fissure bifurcated in a Y-shaped fashion on transverse section. One half-cord then rapidly resumes an almost normal appearance, its central canal persisting as the true central canal of the lower segments. The postero-mesial cornua gradually merge again into the posterior grey commissure. The other half-cord remains for a time as an abnormal excrescence of grey matter projecting laterally from the true anterior cornu. In Fig. 8 the arrangement of the anterior cornual cells is shown. There is a constriction of the grey matter at the point corresponding to the position of the original antero-mesial fissure. On both sides of this constriction there are groups of nerve-cells, the majority being external to the constriction. At this level the central canal of this half-cord becomes less distinct and the posterior cornu disappears (see Fig. 9). The fibres of the posterior root on this side are more numerous, but in smaller bundles, than those of the posterior root of the other side.

In the sacral region a small relic of the additional central canal is still to be made out, situated antero-mesially to the constriction of the anterior cornu. Even in the coccygeal segment there is a trace of an extra posterior septum (see Fig. 11), situated at the circumference of the cord, about a quarter of the way round from the true posterior septum.

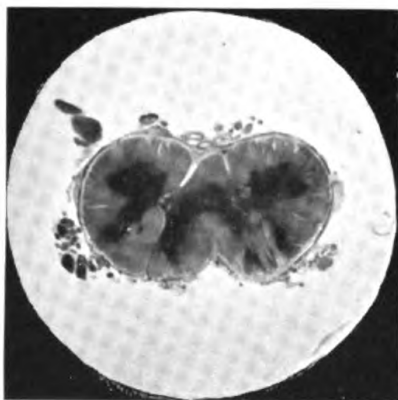
At first sight this case might appear to be an example of true doubling of the spinal cord, the central canal of the original cord bifurcating to correspond to the double organ. But inasmuch as the nerve-cells in the abnormal parts are for the most part, if not entirely, situated in the antero-external cornua, which are the homologues of the anterior cornua of the undivided cord,



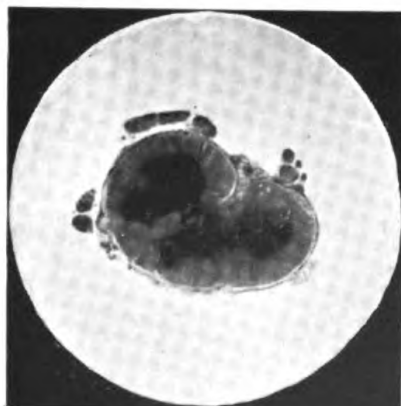
Second Lumbar Segment.  
Photograph  $\times 1\frac{1}{2}$ .



Third Lumbar Segment.  
Photograph  $\times 1\frac{1}{2}$ .



Fourth Lumbar Segment.  
Photograph  $\times 1\frac{1}{2}$ .



Fifth Lumbar Segment.  
Photograph  $\times 1\frac{1}{2}$ .





and considering also that the posterior roots in the bifurcated area are in connection only with the postero-external cornua, it is more probable that the mesial cornua, whether anterior or posterior, are merely masses of expanded grey matter corresponding to the grey matter of the commissure higher up. The expanded grey commissure becomes constricted in the middle by ingrowth of the anterior and posterior median septa with pia mater, and strands of white matter come to separate the mesial cornua from the constricting pia. Moreover, careful examination of the mesial cornua shows their structure to be abnormally diffuse, being traversed by strands of white matter. We therefore have to do, apparently, with a bisected cord, not with a true doubling of the cord.

The literature of this subject has been so recently discussed by Bruce, M'Donald, and Pirie in their admirable article in this Review (1906, part i.) that it is unnecessary to recapitulate the points which they have brought out. We therefore content ourselves with recording this case as another example of the type to which belong the cases recorded by Bruce, M'Donald, and Pirie, by v. Recklinghausen, by Theodor and by Steiner. In all these cases, as in our own, the antero-external cornua were better developed than the antero-mesial. The posterior roots appear to be connected in a similar fashion with the postero-external cornua, the postero-mesial cornua being formed by a diffuse spreading-out of the grey matter of the posterior cornua.

We would direct attention to the presence of pes cavus in our case, a condition doubtless associated with the congenital deformity of the spinal cord.

#### DESCRIPTION OF FIGURES.

*(Drawn to scale with the aid of Edinger's projection-apparatus.)*

- FIG. 1.—First lumbar segment. The spinal cord is single with a somewhat dilated central canal. There is some commencing deformity of the grey commissure in the left side posteriorly.
- FIG. 2.—First lumbar segment, lower than the preceding. The grey commissure has increased in area and shows anterior and posterior outgrowths. On the left side a smaller central canal is present, situated anterior to the original central canal, which is here smaller than in Fig. 1. The anterior and posterior nerve-roots are normal in their positions.
- FIG. 3.—Second lumbar segment. The anterior and posterior septa have deepened to meet each other, splitting the grey commissure into two ill-

defined masses, each possessing a portion of the original central canal. The anterior cornual cells and anterior nerve-roots are normally situated.

FIG. 4.—Second lumbar segment, slightly lower than Fig. 3. The two half-cords are separated by a constriction containing pia mater. On each side an antero-mesial septum is seen. Two complete central canals are present and the grey matter of the commissure is assuming the appearance of anterior and posterior cornua, separated from the surface by a layer of white matter. On the left side, postero-mesially to the central canal, there are a few small nerve-cells. The remainder of the nerve-cells are in the original anterior cornua.

FIG. 5.—Second lumbar segment, lowest level. Half-cords completely separated.

FIG. 6.—Third lumbar segment. Two almost complete cords. The outer portion of each is more complete than its mesial portion. The antero-external cornua contain normally situated nerve-cells. In the antero-mesial cornua only a few poorly developed nerve-cells occur. Two central canals. The nerve-roots, anterior and posterior, are associated with the external cornua only. An antero-mesial septum is present. The two half-cords are rotated so as partially to face each other by their anterior surfaces. On the right side there is a postero-median septum and a rudimentary substantia gelatinosa at the tip of the postero-mesial cornua.

FIG. 7.—Fourth lumbar segment. There is partial fusion of the two halves anteriorly, the posterior portions still remaining separated. On the right side the postero-mesial cornua is poorly defined from the postero-external. The central canals are symmetrical.

FIG. 8.—Fifth lumbar segment. The left half-cord is almost normal, with an anterior and a posterior cornu. Its central canal becomes the main canal of the lower segments. The right half is still abnormal, its anterior cornu being constricted at the point where the small central canal is present. Traces of anterior and posterior septa still persist on this side. The area of the posterior cornu is somewhat indefinite. The posterior root fibres are somewhat scattered. The anterior median fissure is Y-shaped on transverse section.

FIG. 9.—Upper sacral region.

FIG. 10.—Lower sacral region.

FIG. 11.—Coccygeal segment. The asymmetry is less evident but does not entirely disappear. The anterior and posterior median septa resume their normal appearances. Even in the lowest section there is still a trace of an additional central canal. And on the right side, at the circumference of the cord, there is a relic of an additional posterior septum.

# Abstracts

## ANATOMY.

**THE CELL COLUMNS OF THE ANTERIOR CORNUA OF THE (404) SPINAL CORD OF MAN.** (*Les colonnes cellulaires des cornes antérieures de la moelle épinière de l'homme.*) M. et Mme. DÉJERINE, *Rev. Neurol.*, July 30, 1906, p. 689.

DR AND MADAME DÉJERINE showed at a meeting of the Neurological Society of Paris a series of drawings representing serial transverse and longitudinal sections of different segments of the human cord. Their conclusions, which will probably be published later elsewhere, along with the exhaustive details of their research, are that there are great individual variations in the form of the human cord; that the levels of medullary segments may vary not only from one cord to another, but in the two sides of the same cord; that the general contour may show great individual differences, and that the symmetry of the two halves of one cord is rarely complete. In spite of these differences, however, the general characters of different sections are sufficiently constant to allow of one cord being superposed upon and compared with another, and of a characteristic type of contour being recognised in the anterior cornua of each segment or part of a segment.

This conclusion is in harmony with the statement made by the reviewer in his "Topographical Atlas of the Spinal Cord."

ALEXANDER BRUCE.

**ON THE LOBUS CEREBELLI MEDIANUS.** GEIST, *Neurolog.* (405) *Centralbl.*, Sept. 16, 1906, p. 855.

IN two brains Geist found the vermis separated from the lateral lobes of the cerebellum by deep furrows. This condition is regarded by him as an instance of atavism, as the vermis is phylogenetically the oldest portion of the cerebellum and is in the lower mammals distinctly separated from the lateral lobes. This was the only abnormality in the brains; the relative sizes of the different parts of the cerebellum were normal.

GORDON HOLMES.

**NOTE ON THE RETICULATED STRUCTURE OF THE AXIS-(406) CYLINDER.** (*Note sur la structure reticulée du cylindraxe.*) M. G. MARINESCO, *Polytechnia*, Vol. iii., No. 1, 1906.

FROM his researches into the mechanism of regeneration of nerve fibres of the peripheral nerves, Marinesco concludes that there

exists in the normal axis-cylinder of peripheral nerves an extremely fine and slender network, which is rendered more apparent whenever there is a dilatation of its meshes caused by increase of the inter- and peri-fibrillary substance. The greater the quantity of liquid absorbed, the more marked does this network become. The reticulum is pre-formed, and is represented in the regenerating fibres either in the form of a fine network with oblong meshes, or in the form of a network which is coarser, on account of the existence of primary fibres, which are re-united by slender, delicate angles.

These conclusions apply also to the structure of the axone and the axis-cylinder of central neurones. In his experiments Marinesco found that after section of the cord there was present in the central end of the divided medullary fibres a network which could be clearly seen, but which varied greatly in its appearance. Sometimes, by reason of the increase of the intrafibrillary substance, it assumes characteristics which give it a marked resemblance to the network found in nerve-cells. Marinesco's conclusion is, therefore, that there is no essential difference between the structure of the neurofibrils in the cell and in the nerve fibres, so that the anatomical and functional difference which exists between these two organs must lie elsewhere than in the neurofibrils.

ALEXANDER BRUCE

**WEIGERT'S NEUROGLIA STAIN.** (*Zur Technik der Weigert'schen* (407) *Gliafärbung.*) FRITZ HOPPE, *Neurolog. Centralbl.*, Sept. 16, 1906, p. 854.

THE author professes to obtain well-stained neuroglia preparations by mordanting sections of tissue, which have been hardened in formaline and cut in celloidin, in Weigert's chrome-copper mordant for 1 to 3 days at 36° C, and afterwards staining them according to Weigert's original directions. Staining of neuroglia in tissue which had lain for months in 80 per cent. alcohol also succeeded by this method. The advantages claimed for this modification of Weigert's method are saving of time, and the fact that other sections cut from the same block of tissue may be stained by other methods.

GORDON HOLMES.

## PHYSIOLOGY.

**THE DISTRIBUTION OF AFFERENT NERVES IN THE SKIN.** (408) MAX VON FREY (of Würzburg, Germany), *Journ. of Am. Med. Assoc.*, Sept. 1, 1906.

It is agreed by most physiologists that the sensory functions of the skin are based upon four fundamental qualities, giving sensa-

tions of warmth, cold, touch, and pain. Corresponding to these functions, the nerve supply to the skin must be a fourfold one. Blix was one of the first who contended that there were terminal organs of specific function within the skin, whose projection on the surface was indicated by irritable points; von Frey estimates that there are on the skin of the trunk and limbs about 30,000 warm spots, 250,000 cold spots, and half a million touch spots. Reliable determinations as to pain are wanting. Regions are to be found on every normal skin where one or two of the typical sensations are wanting—*i.e.* where cold is felt, but not warmth, pain, but not touch, etc. Von Frey explains this by the "irradiation of stimuli" (physical and physiologic). By physical irradiation he means the spreading of irritation over a larger number of terminal organs than the nature of the stimulus would seem to demand. By physiologic irradiation he refers to the ability to discriminate points as separate on the surface of the skin.

In the perception of pressure a distinction must be drawn between light pressure and deep pressure; light pressure is appreciated by the skin with its touch points; deep pressure by terminal organs called "muscle spindles" (Sherrington).

Concerning the "protopathic" and "epicritic" systems of Head, the author says: "It seems for the present, at least, not necessary to introduce a hypothesis to which existing physiologic and psychologic data do not lend support."

C. H. HOLMES.

## **PATHOLOGY.**

### **THE CONDITION OF THE FIBRILLO-RETICULAR SUBSTANCE**

(409) **IN SWOLLEN NERVE CELLS.** (*Das Verhalten der fibrillo-reticularen Substanz bei Schwellungen der Nervenzellen.*) K. SCHAFFER, *Neurolog. Centralbl.*, Sept. 16, 1906, p. 834.

PRIMARY disease of a nerve cell is characterised by swelling, due to increase of its interfibrillar protoplasm; this is seen after injury to its axis-cylinder, and it is the chief change in amaurotic family idiocy which may be taken as a type of primary cell disease. The swelling may be limited to one part of a cell or to a dendrite, but the axis-cylinder never swells in this way. The author has availed himself of the greater ease with which intracellular neurofibrils can be examined in swollen than in normal cells, as there the fibrils are more definitely separated from one another.

The present descriptions are chiefly from preparations obtained by Bielschowsky's method from the central nervous systems of cases of amaurotic family idiocy. The intracellular fibrils are very indistinctly visible in normal cells, but when a cell is swollen they

are seen to form a network of polygonal meshes, which are smaller in the perinuclear zone than elsewhere. Often the trabeculae of this net can be seen to be continuous in the periphery of the cell with the thicker strands of the pericellular (Golgi) net; and the coarser fibrils which spring from the intracellular net and pass into the periphery of the dendrites also join the pericellular net of Golgi. Thus Schaffer confirms Bethe's assertion that the pericellular net is directly continuous with the intracellular fibrils, a statement which Cajal in chief disputes on the evidence of preparations by his own and other methods. Many of the coarser fibrils of the dendrites can be traced through the cell into the perinuclear zone, where they branch up and become continuous with the trabeculae of the intracellular net.

In further advanced stages of degeneration the intracellular network of fibres becomes disintegrated and its trabeculae break up into a fine granular debris; but different bundles of fibrils are found to be differently resistant to degenerative processes, and the Golgi net may remain intact when the intracellular net has completely disappeared.

GORDON HOLMES.

#### ON DIPLOCOCCUS INTRACELLULARIS MENINGITIDIS AND ITS

(410) RELATIONS TO GONOCOCCI. (Ueber den *Diplococcus intracellularis meningitidis* und seine Beziehungen zu den Gonococcen.) W. G. RUPPEL, *Deut. med. Wchschr.*, Aug. 23, 1906, p. 1366.

THE numerous attempts which have been made within the last few years to obtain a protective and curative serum for use in epidemic cerebro-spinal meningitis have met with little success, one reason being that the virulence of the cultures which have hitherto been available has not been sufficiently constant.

Ruppel has now succeeded in obtaining a meningococcus which, after daily transference to fluid media during a period of five months, was proved to have acquired a high degree of pathogenicity for animals. Intraperitoneal injection of 1 c.c. of a fluid culture in dilution of 1:200,000,000 kills a rabbit in 12 to 18 hours.

After inoculation of horses with those virulent cultures, a serum was obtained,  $\frac{1}{10}$  c.c. of which is capable of protecting a white mouse against one hundred times the minimal lethal dose of virulent meningococci. The serum protects the rabbit against more than 1000 times the minimal lethal dose.

Although the serum agglutinates not only the homologous culture of meningococcus but also all other strains of that micro-organism, the presence of agglutinins in the serum is no proof of its protective or curative efficacy.

The difference in potency of the immune sera obtained after inoculation of horses with virulent cultures of meningococcus, with avirulent strains of the same organism, and with gonococci, is merely a quantitative one.

W. T. RITCHIE.

**ON THE EXTENSION OF ASCENDING MYELITIS.** (Zur Frage (411) über die Wege der aufsteigenden Myelitis.) V. SALLE, *Deut. Zeitschrift f. Nervenheilk.*, Bd. 31, H. 1-2, S. 108.

AN experimental investigation on the paths by which inflammatory conditions spread up the spinal cord. The lower part of the cord (in puppies) was exposed, and various irritants, chemical and bacterial, injected directly into the cord in the region of the central canal. The animals lived from a few hours to several days after the injection. The results varied somewhat with the irritant employed, but in all it was found that the extension was by the blood-vessels (both central and coronal), and also largely by the perivascular lymphatics.

The central canal appeared also to act as a path to some extent, but, although the lesion in its neighbourhood must have been gross compared with that which is present in clinical infective myelitis, its importance seemed slight compared with that of the perivascular lymphatics.

J. H. HARVEY PIRIE.

**THE RESUSCITATION OF THE CENTRAL NERVOUS SYSTEM**  
(412) **OF MAMMALS.** G. N. STEWART, C. C. GUTHRIE, R. L. BURNS, and F. H. PIKE, *Journ. of Exper. Med.*, Vol. viii., No. 2, March 26, 1906, p. 289.

THE object of the experiments, which were performed on cats and dogs, was to gain a knowledge of the condition of the anterior part of the cord and of the brain centres during total acute anæmia, and to determine the ultimate limit at, or below which, resuscitation is possible. Acute temporary cerebral anæmia was produced by passing ligatures around the innominate (from which both common carotids and the right subclavian take origin in the cat) and left subclavian arteries proximal to the origin of the vertebral; traction on the ligature produced occlusion of the arteries. Artificial respiration was used.

The phenomena of complete occlusion are characteristic and constant. The nose and mucosa of the mouth become white as in death, respiration ceases, the reflexes disappear, and the pupils dilate, while the heart is but little affected. Observations were made on the blood-pressure, pulse-rate, respirations, and reflexes



during occlusion and following restoration of the cerebral circulation. For these, which are given in great detail, the original paper should be consulted.

The eye reflexes disappear very quickly, and a period of high blood pressure follows the occlusion; this is succeeded by a fall, then a second rise, and then a slow fall, which is maintained throughout the period of occlusion. Respiration stops temporarily (in 20 to 60 seconds) after the beginning of occlusion, and then follows a series of strong gasps of the Cheyne-Stokes type, after which it stops entirely until some time (it may be as long as one hour) after the restoration of the cerebral circulation. The anterior part of the cord and the encephalon lose all function; no reflexes are obtainable.

Following the return of blood to the brain, convulsions varying in severity, of tonic or clonic type, sometimes begin before the full return of the reflexes, or occasionally the animal may lie quiet from 1 to 3 hours before their onset. These spasms, which are always present, may terminate in death or in partial or complete recovery. Transection of the cord stops the spasms below the level of the section, and hemisection stops them on the side of the section below its level.

Death, without any return of the reflexes after release of the cerebral arteries, has followed an occlusion of  $7\frac{1}{2}$  minutes. Respiration has returned after an occlusion of 1 hour. Five animals recovered completely after an occlusion of 7 minutes or more; only one animal recovered completely after an occlusion of 15 minutes, and none after 20 minutes. Beyond this period of complete cerebral anæmia the authors believe that resuscitation is not possible.

SUTHERLAND SIMPSON.

#### **EXPERIMENTAL CEREBRAL ATROPHIES AND ACCOMPANY-**

(413) **ING CRANIAL ATROPHIES.** G. D' ABUNDO, *Annali di Neurol.*, Anno xxiii., f. vi.

FROM an earlier series of experiments, which were carried out on cats and dogs twenty-four hours after birth, the author was enabled to arrive at the following conclusions with regard to the production of cerebral hemiatrophies: (1) ablation, more or less extensive, of the vault of the cranium, without injuring in any way the cerebral substance, does not lead to any hemiatrophy of the brain; (2) removal of the cortex cerebri down to the white matter produces cerebral and cranial hemiatrophy, even when the ablation of the cranial vault is quite limited. In all these cases the cranial atrophy was considered to be secondary to the lesion of the corresponding hemisphere.

In this paper we find the results obtained by producing similar lesions of the cortex in both cerebral hemispheres of new-born cats and dogs.

The result of these experiments was to give rise to a condition of atrophy of both cerebral hemispheres, together with a corresponding absence of growth of the cranium; and if in any case one hemisphere was atrophied more than the other, the absence of cranial growth on that side was also more marked. In all these cases the development of the whole of the body of the animal was somewhat disturbed, and in those which lived long the condition of ventricular hydrocephalus appeared. The asymmetry produced if the operation was done when the animal was thirty days old was much less than when it was performed twenty-four hours after birth. In adult animals removal of the vault did not affect the brain, nor did the removal of the cortex produce any change in the cranium. In these latter cases the condition of hydrocephalus "ex vacuo" always accompanied the atrophy of the brain.

It is established, therefore, that cerebral morbid processes which act on the brain in the earliest period of extra-uterine life have a most important influence on the conformation and on the development of the hemisphere operated on, and also on the corresponding part of the cranium; that the cerebral lesion determines the cranial asymmetry; and that the earlier the morbid process acts, the more serious will be the cerebral and cranial disturbance. It is probable that the more serious anomalies which are met with in idiots are the result of morbid processes which were active during intra-uterine life.

It is necessary also, from the clinical and medico-legal point of view, to remember that very serious results may follow an injury to the head in the earliest days of extra-uterine life, and that a morbid cause which interferes with the normal function of a cortical zone at an early period, when the brain is developing rapidly, limits the evolutive potentiality, not only of the cerebral hemisphere corresponding to the lesion, but also of the entire brain.

R. G. Rows.

**CONCERNING THE EFFECT OF EXPERIMENTAL SECTION OF**  
(414) **THE POSTERIOR ROOTS UPON THE PERIPHERAL**  
**NEURONS.** (De l'influence de la section expérimentale des racines postérieures sur l'état des neurones périphériques. Contribution à l'étude des fibres centrifuges des racines postérieures.) ROUX et HEITZ, *Nouv. Icon. de la Salpét.*, July-August 1906, p. 297.

In the first weeks after section of the posterior roots, the cutaneous nerves and mixed trunks always show a slight degree of

**Wallerian degeneration.** The same degenerative process affects the sympathetic, is evident at the third week, and seems to stop by the eighth and eleventh month. The coarse fibres are preserved; these only degenerate after ablation of the spinal ganglia. For studying the changes in the spinal ganglia the authors prefer the osmic acid and sublimate method, followed by carmine, to Nissl's, as the fibres are stained as well as the nerve cells. The cells were found well preserved, but some fibres, especially at the central end of the ganglion, were in parts swollen, at others thinned, with irregularity of outline.

In the ganglionic end of the posterior root the appearances varied with the date of examination. At the fifth day a certain number of fine fibres were degenerated, and by the 247th day the large fibres were thinned, badly coloured, irregular in outline, and fragmented in places, but not disintegrated into droplets. A large number of fine fibres were present, giving one the impression of regenerated fibres.

In the medullary end of the sectioned posterior root very fine normal fibres were found, and in the ganglionic stump a corresponding number of degenerated ones which could be followed in the afferent nerve from the ganglion almost as far as its fusion with the anterior nerve (fifteenth day, third month). These are regarded as centrifugal fibres.

At the 382nd day the medullary end showed a very large number of fine fibres, probably coming from the spinal ganglion cells.

The authors then proceed to discuss what the influence of posterior root section is upon the peripheral neurons, and what deductions can be drawn with regard to the pathogenesis of tabes.

In the cutaneous branches, at the fifteenth day, the degeneration is of fine centrifugal fibres, but, by the 250th day, medium-sized fibres are also involved. This is ascribed to a peripheral neuritis of the Wallerian type, affecting exclusively the fine cutaneous branches. By the time this neuritis has appeared alterations are found present in the ganglionic end of the sectioned root, while the ganglion cells remain normal.

By the end of the first year the peripheral neuritis disappears, leaving the nerve sheaths empty, while retrograde degeneration of coarse fibres and fine regenerated fibres can be found.

These latter are prolonged into the medullary stump. Thus some fibres showed degeneration of both prolongations, Wallerian of the peripheral end, simple atrophy of the central portion.

**Conclusions:**—In the posterior roots of mammals there are myelinated centrifugal fibres, relatively few in number, and mostly of fine calibre. They pass for the most part into the sympathetic system, by the rami communicantes. A small number pass into

the peripheral nerves. At the seventh and eighth months after root section the cutaneous nerves show Wallerian degeneration, and to a much less extent the mixed trunks also; the ganglion cells are normal, and the ganglionic end of sectioned posterior root shows retrograde degeneration. A year after operation, regeneration has taken place in the medullary end of the cut root.

The authors recognise the difficulty of drawing a parallel between experimental work such as theirs and the results of root affection in tabes, and point out many differences, *e.g.* the affection of ganglion cells and different type of fibre degeneration in tabes.

They think that, besides the direct action of meningitis on the posterior roots, a certain part of the disease may be due to the toxic influence exercised by the syphilitic virus. This toxic action is manifested mainly on the sensory proto-neurons and the centripetal proto-neurons of the sympathetic.

DAVID ORR.

**NEUROTROPISM AND TRANSPLANTATION OF NERVES.** (Sul (415) *neurotropismo e sui trapianti dei nervi.*) LUGARO, *Riv. di Patol. Nerv. e Ment.*, F. 7, 1906, p. 320.

IN an earlier series of experiments the author observed—after cutting both anterior and posterior nerves at the same level and removing the spinal ganglion—that regenerated fibres passed from the central stump of the anterior nerve into that of the posterior. This phenomenon might be explained by the theory that the regenerative products in the posterior roots diffuse substances which exert a positive chemotropism on the new axis-cylinders of the anterior roots and draw them into the empty nerve sheaths. Once having gained these, the new fibres push their way along the posterior root as far as the cord surface, but not into its substance. They infiltrate the pia mater, both outside and inside the point of root entry, and spread upwards and downwards for some distance on the cord surface. Before discussing the source of this neurotropic action, Lugaro refers to the statements of Bethe on polarity of nerve fibre. The latter admits that all fibres have a certain polarity, not exercised in the direction of the nerve current, but in that of the orientation of the fibre from its cell; so that in peripheral nerves the sensory and motor fibres are traversed by nerve currents of opposite direction but have the same polar orientation.

In Lugaro's experiments, therefore, the new fibres in the central end of the motor nerve find the correct polarity in the central end of the posterior root, which is separated from its trophic centre, and so equivalent to the peripheral end of a nerve. The cells of Schwann's sheath exert a neurotropic action on the new axis-cylinders, which allows of their invasion of the posterior root, but

directly these cells cease to exist the axis-cylinders are repelled at the cord margin by a negative neurotropism, and, as above indicated, infiltrate the pia mater.

As confirmatory evidence of the above observation, the author conducted the following experiment:—Both seventh lumbar nerves were cut outside the spinal ganglion; this was then removed (both sides), and the anterior nerve of the right side united to the posterior nerve of the left side and *vice versa*. The experiment was successful, and confirmed the views already expressed regarding the positive neurotropism of the cells of Schwann and the negative neurotropism of the central nervous system.

To study the behaviour of the axis-cylinders of the central nervous system in the presence of Schwann's cells a piece of sciatic nerve was embedded in the brain of the same animal. Examined by Cajal's reduced silver method, the embedded nerve showed no new formed axis-cylinders, thus showing that Schwann's cells exercise a neurotropic action on the axis-cylinders of peripheral nerves which are regenerating, but not on those of the central nervous system.

Two questions are now discussed. Can this neurotropic action make itself felt on a normal nerve enveloped in its normal sheath; and from where do pieces of nerve embedded close to other nerves derive their fibres?

Having embedded pieces from one sciatic close to that of the other side (same animal), Lugaro found these, at the fifteenth day, adherent to the muscle, but not to the normal sciatic. There was no sign of penetration by young fibres and the sound sciatic was quite normal.

At the twenty-sixth day the embedded nerve was found firmly adherent to muscle, from which non-medullated fibres sprang, penetrating first the cicatricial tissue, then extending between Schwann's nuclei. The sciatic was absolutely normal. The author concludes, therefore, that embedded nerves do not exert any neurotropic action upon normal nerves in their vicinity. That a process of auto-regeneration does not take place is shown by the fact that the fine axis-cylinders entering the embedded nerve are perfectly continuous and end in a fine olivary swelling, thus showing them to be in a condition of active growth and running isolated in a large collection of chains composed of Schwann's cells perfectly empty.

To confirm these experiments, Lugaro extirpated the spinal ganglion with the adjoining part of the posterior root without inflicting the least injury upon the anterior one. Here he found that the anterior root gave off no axis-cylinders to the stump of the posterior one.

Even where a degenerated fasciculus of a nerve runs side by side with a normal one in a common sheath no transverse neuro-

tropic action is provoked. Probably neurotropic action is diffused only from the central end of a divided nerve, and does not influence normal fibres. Its influence is greatest on the central stump of divided fibres, which are growing.

*Conclusions:*—1. Fibres from the anterior root can be continuous with those of the central stump of the posterior root. 2. They follow the normal path so long as there are cells of Schwann, then penetrate the pia mater. 3. The central nervous system exercises a negative neurotropism on the anterior root fibres. 4. The cells of Schwann, the origin of neurotropism in the regeneration of peripheral nerves, do not exert any neurotropic action on the axis-cylinders of the nerve centres. 5. Transplanted nerves in the vicinity of a sound nerve, whose sheath is uninjured, have no action upon it. 6. They become adherent to muscle, and draw from it nerve fibres which, by neurotropic action, are drawn amongst the cells of Schwann.

DAVID ORR.

#### **RETROGRADE DEGENERATION IN THE SPINAL NERVES.**

(416) S. W. RANSON, *Journ. of Neurol. and Psychol.*, Vol. xvi., No. 4, 1906.

IN this excellent piece of work the author begins with a historical account of retrograde or ascending degeneration.

The writer collates the results of researches conducted by many workers under certain headings. These include the changes in the central end of a divided nerve, in the anterior and posterior roots belonging to the nerve in question, in the spinal ganglia, and in the spinal cord, including the anterior and posterior horns and the posterior columns of the cord. The material from which the historical part of the work is drawn consists of sixty-nine autopsies of amputation cases in the human subject, and the experimental researches on animals of eighteen observers. The author finds, partly from the work of these other investigators, and partly from his own experiments on rats, that simple atrophy and also true degeneration result from nerve section. These changes are seen in the central end of the divided nerve, in the spinal ganglia, in the posterior and anterior nerve roots, and in the spinal cord. The atrophy causes a decrease in size of the nerve fibres, some of which lose their medullary sheaths, while many of the nerve-cells of the anterior horn and of the spinal ganglia also undergo atrophy. The degeneration begins in the central end of a divided nerve some weeks later than the Wallerian degeneration in the peripheral segment, but corresponds closely with it in microscopic characters. This degeneration is limited as far as the fibres in the nerve are concerned, but it extends into the cord. The

anterior cornual cells undergo a certain degree of degeneration, some of them disappearing altogether, while a considerable and more constant number of the spinal ganglia cells also vanish.

It is well recognised that there are far more cells in the spinal ganglia than there are nerve fibres in the posterior nerve roots corresponding to them; this implies that many of the cells and probably the smaller ones are not connected with nerve fibres in the dorsal roots at all. As the result of section of all the fibres passing to the spinal ganglion of the second cervical nerve in the rat, the author found that more cells in the spinal ganglion degenerate than can be explained by the division of the nerve fibres. In the posterior nerve root after section of the nerve there was a variable disappearance of fibres, but equalling an average of 17 per cent. Ranson notes that the dorsal nerve roots tend to degenerate more in young than in adult animals.

The statement that the degeneration of the spinal ganglion cells was constant after section, while the number of nerve fibres in the posterior nerve roots varied, constitutes an important part of the paper, although no explanation is offered with regard to the discrepancy. We are pleased to find that the author corroborates the statement that the degenerative changes following on nerve section cease after a period of two months.

The greatest care was taken to prevent any sepsis, during or after the experimental operations, and in the enumeration of the nerve cells those cells only were counted whose nucleoli were clearly seen, a method which should ensure accuracy in counting where several thousand cells had to be enumerated in each ganglion, and numerous serial sections had to be examined in reckoning the cells even in the comparatively small ganglion of the rat. A very prudent precaution was adopted, namely to take as control enumerations the same spinal ganglia from animals of about the same age, but which had not sustained any operation.

ROBERT A. FLEMING.

## CLINICAL NEUROLOGY.

**COEXISTENCE OF TERTIARY SYPHILITIC LESIONS WITH (417) TABES AND GENERAL PARALYSIS.** (*Coexistence d'accidents syphilitiques tertiaires avec le tabes et la paralysie générale.*) DORLEANS, *Thèse de Paris*, 1906.

DORLEANS has collected twenty-eight cases in which tabes or general paralysis was associated with tertiary lesions of the skin, bones, tongue, eye, brain, and testis.

In nearly half the cases the patients denied or were unaware

of the existence of syphilis. The author's conclusions are as follows:—

1. The coexistence of tertiary syphilitic lesions with tabes or general paralysis is not rare.
2. This coexistence is of interest, because it affords a further proof of the syphilitic nature of tabes and general paralysis, and may help to an early diagnosis, and to the establishment of a rational and efficacious treatment.

J. D. ROLLESTON.

**THE RELATION OF SYPHILIS TO LYMPHOCYTOSIS OF THE  
(418) CEREBRO-SPINAL FLUID AND TO THE QUESTION OF  
"MENINGEAL IRRITATION."** (Die Beziehung der Syphilis zur Lymphocytose der Cerebrospinalflüssigkeit und zur Lehre von der "meningitischen Reizung.") L. MERZBACHER (of Heidelberg), *Centralbl. f. Nervenhe. u. Psych.*, July 1, 1905, May 1, 1906.

In this contribution Merzbacher follows up the work of Nissl (*vide abstract, Rev. of Neur. and Psych.*, Vol. ii. p. 479), who took a stand against the rather loose pathogenic views of the French school with regard to lymphocytosis of the cerebro-spinal fluid, and defined the exact state of our knowledge of the subject. He said "to my mind it is not at all definitely established that a positive cytological result is of necessity due to an inflammatory process in the meninges." Merzbacher endeavours to answer two questions. (1) How far can syphilitic infection produce a lymphocytosis without there being any clinically demonstrated disorder of the central nervous system and its membranes? (2) Must every lymphocytosis in the syphilitic be attributed to the "meningeal irritation" of the French school?

In order to answer the first question, he examined the available material in the Heidelberg clinic, choosing patients with a definite history of syphilis, but with no organic affection of the central nervous system.

In 89·7 per cent. of the cases the result was positive. The number of cells found was much less than that usually found in cases of tabes and general paralysis. The conclusion is that in almost all his cases syphilitic infection had led to an increase of the cell elements in the cerebro-spinal fluid, even in the absence of any evidence of disease of the central nervous system and of the meninges.

He begins his attack on the second question by examining the results of others with regard to a lymphocytosis in cases with disorders of the pupil movements, but who were neither tabetics nor general paralytics. He agrees with Bumke that the Argyll



Robertson phenomenon as an isolated sign is not necessarily due to a syphilitic meningitis, but depends certainly on syphilitic infection. Many cases present this sign who do not later become either tabetic or general paralytic.

There is no sound reason for explaining the lymphocytosis which uniformly accompanies the Argyll Robertson pupil by a local affection of the meninges. The latter is purely hypothetical. In cases of eye disease of various nature, where there was no reason to suspect the presence of meningeal disorder, there was always a history of syphilis in the cases where the puncture gave a positive result. Similarly in cases of brain tumour, hemiplegia, paraplegia, and cerebral apoplexy the cases which presented a lymphocytosis differed from those which failed to do so, not in local differences of the process, but in the presence of a history of syphilitic infection. Must one attribute the lymphocytosis in tabes and general paralysis to the meningitis present? The lymphocytosis is present and even most abundant at an early stage in tabes when there is no evidence that there is a meningitis. Meningitis, according to some, is not always present, and in many cases it is a purely hyperplastic form (Nissl). In general paralysis the characteristic plasma-cells so common in the meningeal exudate are not found in the cerebro-spinal fluid, at least to any extent. Even in tabes and general paralysis it is safer to refer the lymphocytosis to the syphilitic infection than to the meningeal changes, although the latter do no doubt play later an important part in producing the lymphocytosis. Other infections produce lymphocytosis without there being evidence of a meningeal factor—multiple sclerosis, herpes, mumps.

Merzbacher concludes that the theory of meningeal irritation lacks evidence; he finds the common factor of all cases with lymphocytosis in the syphilitic infection, which in some way disturbs that mechanism, which even under physiological conditions allows some cells to reach the cerebro-spinal fluid.

C. MACFIE CAMPBELL.

**A CASE OF AMYOTROPHIC LATERAL SCLEROSIS.** (*Un cas de (419) sclérose latérale amyotrophique.*) PUSCARIN et LAMBRIOR, *Rev. Neurol*, Sept. 15, 1906, p. 789.

A RECORD of a pretty typical case in a man of forty. Commenced in the fingers of the right hand with some numbness, then muscular atrophy; rapidly extended up the arm, at the same time the left hand and arm becoming involved. The lower extremities then became involved, and all were characterised by marked rigidity and contractures.

In less than five months the muscles of the neck, head, and face were affected ; swallowing became impossible, respiration irregular, and death occurred within seven months of the onset.

Microscopically there was found a marked degeneration and sclerosis of the crossed pyramidal tract, from the cerebral peduncles to the sacral cord ; the direct pyramidal tract being less severely involved. There was also some sclerosis of the whole antero-lateral columns. There was typical degeneration of the anterior motor-cells, at its maximum in the cervical enlargement ; also of the hypoglossal, vagus, and facial nuclei.

No light is thrown on the etiology of the condition.

J. H. HARVEY PIRIE.

**TRANSVERSE MYELITIS AS A SEQUELA OF MEASLES.** (Un (420) caso di mielite lombare transversa consecutiva a morbillo.) PRIMANGELI, *Il Policlinico*, Sept. 9, 1906, p. 1161.

MEASLES, the commonest of all diseases, is seldom followed by grave nervous sequelæ. Primangeli's case is of special interest in that it was that of an adult in whom a circumscribed portion only of the spinal cord was involved.

A robust countrywoman, aged 25, free from tubercular or syphilitic taint, had an attack of measles. The eruption was of short duration, and the febrile period did not last more than six days. Two days after getting up she was seized with pain in the lumbar region, radiating to the abdomen and lower limbs. She had an urgent desire to micturate, but complete retention was present. The next day spasm of the anal sphincter developed, and paresis of the lower limbs. The temperature was 102°.

There was diminution of tactile and painful sensibility of the lower limbs and of the abdomen to a little above the umbilicus. Some areas of the lower limbs were affected with anæsthesia dolorosa. Active movements of the legs, which had become somewhat œdematous, were very limited. There was diminution of the superficial reflexes, and of the knee- and ankle-jerks. Electrical muscular excitability was impaired.

The patient complained of girdle pain, and of formication and numbness in the lower limbs. There were no motor nor sensory changes in the upper limbs, trunk, or face. After about a week, complete anæsthesia and flaccid paralysis of the lower limbs supervened, with total abolition of the reflexes and incontinence of urine and fæces. The muscles rapidly atrophied, the skin became dry and rough, and in spite of the greatest attention, large bed-sores formed on the sacrum and gluteal regions. Cystitis, hypo-

static pneumonia, and gastritis subsequently occurred, and the patient died after an illness of six and a half months.

The autopsy, of which no details are given, revealed softening and degeneration of the lower dorsal and lumbar cord.

J. D. ROLLESTON.

**A REMARKABLE CASE OF POTT'S PARAPLEGIA.** (*Un cas* (421) *remarquable de paraplégie pottique.*) H. BOSCHI et A. GRAZIANI, *Rev. Neurol*, Sept. 15, 1906, p. 799.

THE patient, a lad of sixteen, woke up one morning—having been perfectly well previously—with tingling and feeling of heaviness in the lower limbs, and found that they were almost quite paralysed. There was severe abdominal pain, which, however, soon subsided and never returned; hypoesthesia for touch and pain from waist downwards; and incontinence of urine and faeces. No pain along the spine, and no elevation of temperature.

When examined by the authors a fortnight later there was found a slight dorsal scoliosis, and some pain, on heavy pressure, over the fourth dorsal spine. No pain on movement of the spine. Voluntary movements of lower limbs very feeble. Cutaneous reflexes diminished, tendon reflexes more active than normal, ankle clonus and double Babinski sign.

Hypoesthesia for touch and pain in the feet and muscular sense defective in the lower limbs.

Micturition and defaecation as at onset.

The cerebro-spinal fluid contained a few lymphocytes and a considerable quantity of albumen.

His condition steadily improved, and three months later there was complete control of the sphincters; the power of movement was very much better. The reflexes were much as before, although less removed from normal; Babinski sign still present. The pain on pressure over fourth dorsal spine was almost entirely gone.

Although a diagnosis could not be made with absolute certainty, the authors consider that the most probable cause was a limited Pott's disease with some associated pachymeningitis.

J. H. HARVEY PIRIE.

**PNEUMOCOCCAL CEREBRO-SPINAL MENINGITIS AND DIABETES.** (*Méningite cérébro-spinale à pneumocoques et diabète.*) LOUIS-ALBERT AMBLARD, *Arch. Gén. de Méd.*, Sept. 11, 1906, p. 2319.

THIS is the record of a case in support of the theory that certain forms of diabetes are of nervous origin. The patient, a man aged 55, was admitted to hospital on the tenth day of an acute

pneumonia, no crisis having occurred. His urine contained a trace of albumen but no sugar. In a few days his symptoms had subsided, but about a week later a rise of temperature and pulse occurred, accompanied by arthritis of the left wrist. This was followed by paresis of the right side and other signs indicating meningitis. The patient died within two days. Immediately after the onset of the meningitis the urine was found to contain sugar to the amount of 154 grammes per diem. The autopsy revealed an unsuspected encysted empyema, a meningitis particularly marked at the base of the brain, and nothing else worthy of note except that the pancreas was healthy. The writer considers the case one of genuine diabetes due either to infective or nervous influences, and pronounces strongly in favour of the latter on account of the time of appearance of the sugar, and the history of the case.

HENRY J. DUNBAR.

**THE SENILE BRAIN.** (*Le cerveau sénile.*) ANDRÉ LÉRI, *Revue* (423) *Neurol.*, Aug. 30, 1906.

IN a résumé the author draws a distinction between old age and senility—the latter being a pathological condition which may make its appearance at any age, late or early, and may affect the organism as a whole or in part.

The pathological changes due to senility are of three special types:—

- (1) Atrophic changes in parenchymatous tissues.
- (2) Proliferation of interstitial tissues.
- (3) Sclerotic lesions in blood-vessels.

The rest of the paper consists of an anatomical and a clinical study of senility.

Macroscopically the brain is small and atrophied, a change shared by the meninges and the central nuclei. The grey matter seems less affected than the white. The ventricles are frequently dilated and their walls thinned.

Microscopically the nerve-cells are diminished in number, and show degenerative changes consisting of destruction of Nissl's granules, and an increased yellow pigmentation.

These changes closely resemble those due to chronic intoxications (alcoholism, uræmia, etc.), and are not peculiar to the senile brain. The nerve-fibres share in the general atrophy, especially the tangential fibres of Tuczak, a change comparable to that found in cases of dementia. The neuroglial cells and fibres are proliferated to a moderate degree, determining a diffuse sclerosis, which, however, is most marked in the perivascular areas.

Sclerotic changes occur in the vessels, but vary much, the

commonest type being one which involves the whole of the arterial wall. The vascular lesions determine the local changes which are met with, such as perivascular sclerosis, miliary hæmorrhages, areas of lacunary degeneration, softening, and cerebral hæmorrhage. Especially typical of senile brain are lacunary degeneration and what the author describes as the "worm-eaten state," due to progressive degeneration of cerebral tissue with neuroglial proliferation.

Clinically the senile changes find expression in an "intermittent claudication" of the brain—symptoms of which are vertigo, headache, tinnitus, sleepiness or insomnia, transitory aphasia, and hemiplegia. Hemiplegia other than transitory as a true senile condition is not very common, and when found is chiefly due to lacunary degeneration, and the lower limb is chiefly affected.

Senile epilepsy may occur, and much more rarely paraplegia of cerebral origin.

The mental condition varies widely from slight changes in memory, character, and intellectual power to simple dementia, which, however, is often slighter than appears at first sight—the patient remaining conscious of his condition for a long time. Signs of general arterio-sclerosis (cardiac, renal, etc.) are always to be found associated with the cerebral lesions.

One must always distinguish between mental changes of old age and those which may occur in old age.

The author concludes in favour of senility being the result of chronic toxic processes.

C. M. HINDS HOWELL.

## **TWO CASES OF PONTINE HÆMORRHAGE. HYPERPYREXIA.**

(424) **RAPIDLY FATAL TERMINATION.** (*Deux cas d'hémorrhagie protrubérantielle. Hyperthermie. Mort rapide.*)  
PIERRE MARIE and F. MONTIER, *Nouv. Icon. de la Salpét.*,  
July-Aug. 1906, p. 383.

THE first case was that of a man aged 86, whose illness began suddenly with vomiting followed rapidly by unconsciousness. He showed a complete left hemiplegia with a certain rigidity of the right side and turning of the head to the right side. He died two days later, the temperature shortly before death being 105·4°. Post-mortem an extensive hæmorrhage into the right side of the pons was found. The second case occurred in a man aged 47, who died the day after the onset of a left hemiplegia associated with rigidity of all the limbs, more marked on the right side, and pronounced myosis. The temperature before death reached 109°. At the autopsy a small hæmorrhage on the right side of the pons

was discovered. In a case of hemiplegia with an apoplectic onset, a rapid course, and accompanied by myosis and hyperpyrexia, one should always be strongly suspicious of a lesion of the pons.

HENRY J. DUNBAR.

**DIVER'S PALSY.** (*La maladie des scaphandriers.*) BOINET, *Arch. (425) Gén. de Méd.*, Sept. 11, 1906, p. 2305.

THIS is a continuation of a previous article of which an abstract appeared in these columns (*Rev. of Neurol and Psychiat.*, 1905, p. 805).

The cases described have been met with amongst the divers employed in the sponge and coral fisheries of the Mediterranean for the most part. A number of fatalities, however, have occurred in the course of salvage operations on the wrecks of sunken vessels on the French coast.

The illness of divers, like that of caisson workers, is due to too rapid atmospheric decompression. Both of these form a chapter in compressed air illness. There have, just recently, been several fatal cases and a number of cases of incurable paralysis amongst the divers off the coast of Provence. On account of all these accidents, which are mostly preventable, it is desirable, the writer thinks, that the apparatus used by the divers should be properly inspected, and that prophylactic measures should be enforced.

The clinical history of fifteen cases, of which seven proved fatal, is given in detail.

In each instance the symptoms appeared after the diver had ascended, *i.e.* on decompression. Death in some cases was sudden, and occurred as soon as the diver reached the deck of the boat. In other cases the patient passed into a state of coma and died after a period of hours, or sometimes days. The fatal issue was usually preceded by "pains" in the limbs, and in the case of those patients who did not die suddenly, by paralysis of the lower limbs and retention of urine. Paralysis of the arms, when it occurs, is usually transient. Cyanosis was observed on some occasions, and râles indicating pulmonary congestion were heard in the majority of fatal cases. All the fatal cases are attributed by the author to too rapid decompression.

Of the non-fatal cases, the typical variety is paraplegia with sensory disturbances. At the onset of the illness there may be paralysis of the arms, but this usually passes off rapidly. In those cases of paraplegia which are permanent, the paralysis becomes spastic in character. At first there is retention of urine and fæces, and later on imperfect control of the sphincters. Anæsthesia of the legs is present, with hyperæsthesia often in the early stages.

Many of these cases have been under the care of the author for a few years, and are regarded as incurable. In rarer cases hemiplegia, with aphasia, is met with, the latter being sometimes temporary, but occasionally permanent. The paralysis in one or two instances takes the form of a monoplegia, either of the arm or leg. A few of the patients had anæsthesia of the palate.

In accidents due to rapid decompression, ecchymoses under the skin, often of the chest, are seen.

Hæmatomyelia is the condition which the author believes to be the cause of the serious symptoms of diver's palsy. The lesions affect the whole of the spinal systems, with the exception of the anterior horns, and do not pass above the level of the second dorsal segment. The presence of hæmorrhages in the cord has been verified both by autopsies on the human subject and after experiments on animals. There are also present in such cases hæmorrhages in the spinal meninges. It is believed that the hæmorrhages in the spinal cord are produced in two ways. In some cases the bubbles of gas liberated in the blood by too rapid decompression actually cause rupture of the capillaries in the substance of the cord. In others, hæmorrhagic infarction, following embolism of a vessel of the cord resulting from bubbles of gas, is thought to occur. The author adheres to the theory of the pathology of the disease attributed to Paul Bert. Nitrogen is dissolved by the blood in increased quantity at high pressure, according to Dalton's law, and is liberated in the form of bubbles on decompression. In experiments the formation of these bubbles can be actually watched. Oxygen poisoning has been suggested as a factor in ætiology, but the atmospheric pressure is not sufficiently great in diving or in caissons for oxygen to exert its toxic effect. The air-embolism theory finds support from the good results of slow decompression and from the therapeutic action of recompression, followed by very slow decompression, in cases of illness. One of the author's patients, who was the subject of paraplegia, showed a marked improvement after dives taken at moderate depths and followed by a slow ascent. Slowness of decompression is the basis of true prophylaxis. In caissons the time given for coming out is, as a rule, three minutes per atmosphere. In the case of divers, the conditions of work do not allow of such a long period of decompression. However, the ascent should be made slowly enough to give at least one minute per atmosphere. In practice, the divers after descending to a depth of forty metres (four atmospheres) usually come to the surface in less than one minute. Such has been the case in all the fatal cases. The result of this rapid decompression is that bubbles are liberated in the capillaries of the lungs and central nervous system. Thus is explained the sudden death, which in certain cases resembles that due to the entrance of air into the veins. The

diving suits in use are often faulty and in bad repair. The supply of compressed air is consequently irregular and its pressure not accurately indicated by the manometer. The absence of rigidity in the old type of suits exposes the wearer to injurious pressure. As a consequence, it is necessary to send air to him at an excessive pressure when he is working at the greatest depths. A new diving suit (Buchanan-Gordon) has been devised to overcome these defects. The body and arms are protected from pressure by spirals of metal, and the diver is enabled to breathe air at a pressure nearer normal.

The author remarks, in conclusion, that better apparatus and a more rigorous application of prophylactic measures, of which the chief is slow decompression, would prevent most of the accidents due to compressed air.

ALFRED PARKIN.

**CEREBRAL AND OPHTHALMIC COMPLICATIONS IN SPHENOIDAL SINUSITIS.** ST CLAIR THOMSON, *Brit. Med. Journ.*, Sept. 29, 1906.

THE author thinks that, if the accessory sinuses of the nose were systematically examined at all post-mortems which showed intracranial inflammatory lesions, we should find that a large number of these was due to suppurative sinusitis.

Observations have shown sinus disease to exist in 30 per cent. of all post-mortems (Harke, Fraenkel, and others); next to the maxillary antrum the sphenoidal sinus is most often affected, and Lermoyez observes "sphenoidal suppuration is not rare; it is only its diagnosis which is uncommon."

Two cases are recorded which have recently come under the author's observation.

Case I. Male, age 36, had two years' history of ear trouble; one month before admission to hospital had influenza and acute earache. On admission he gave history of discharge from nose running chiefly into back of throat, "as long as he could remember." Occasionally severe headaches and bad taste in his mouth; the headache had been chiefly frontal, beginning at 9.30 A.M. and ceasing at 3.30 P.M.; recently the headache had been referred to the lower occipital and left temporal region. The patient looked very ill, pulse 92, temperature 98.8, pupils equal and reacting normally; no vomiting, rigors, or staggering. Left ear showed a perforation of the membrana with slight non-fetid discharge. The right middle turbinal was enlarged and there was pus on the roof of the naso-pharynx on the right side.

The patient did not sleep well, and complained of pain behind



the eyes and on the top of his head, and also of a rotten-egg taste from the back of his nose; he rapidly became dull and irrational.

Operation was decided against as a diffuse meningitis had already started; later diplopia, left internal strabismus, nystagmus and deafness developed; the optic discs were slightly hazy. Before death, facial paralysis was noticed on the right side.

*Post-mortem.*—Pus in right sphenoidal sinus only. Lining membrane of this cavity was thickened, purplish, and sodden. All other sinuses were healthy. Basal meningitis was present, extending down the spinal cord. The author thinks the infection must have been through the lymphatics.

Case II. Female, age 16, had pain on the vertex followed by sickness, shivering, and swelling of the eyelids. A week later the eyeballs were prominent with chemosis of the right conjunctiva. Temperature 102°-105° F. No history of nasal discharge or headache. Stiffness of the neck and backache were next complained of. On admission to hospital, optic neuritis found to be present. Ears quite normal. The nose showed pus on the right side in the middle meatus and olfactory cleft. The patient was quite rational.

The leucocytes numbered 36,000, and the patient had two rigors within 24 hours of admission. Temperature 106° F. Anæsthetic, right middle turbinal removed and right sphenoidal sinus opened and scraped; thereafter irrigations of the sinus carried out twice daily. Later the proptosis and swelling of the eyelids increased and the sixth nerve was paralysed on the right side. Pus, obtained on incising the conjunctiva, showed a pure streptococcus.

*Post-mortem.*—No meningitis. Cavernous petrosal and lateral sinuses all filled with pus. Superior longitudinal normal. Mucous membrane of right sphenoidal sinus thickened and polypoid. Left sphenoidal sinus small and contained recent muco-pus.

The author has collected 42 cases in which death occurred from intracranial complications due to septic infection from the sphenoidal sinus. A list of these, in chronological order, is appended to his paper. In 17 meningitis was the predominant lesion; in 4 thrombosis of the cavernous sinus was the primary complication; and in 13 there was both thrombosis and meningitis. There was only one case of brain abscess.

The chief symptom of sphenoidal sinusitis is post-nasal discharge with anosmia or cacosmia. Unfortunately the discharge may not always be present, and thus the disease may be overlooked unless the middle turbinal be removed and the sinus washed out.

Pain or neuralgia may be referred to the frontal, occipital, or temporal regions. Pain deep in behind the eyes is characteristic

of sphenoidal disease. The pain may be referred to one or the other accessory sinuses, and these cavities and also the mastoid process have, for this reason, been operated on in error.

Sphenoidal sinusitis causes more disturbance of the general health than fronto-maxillary suppuration.

The bacteriology of these cases is briefly mentioned, and the paths of infection are given as follows: (a) directly through the bone; (b) by the veins; (c) by the lymphatics. The roof of the sphenoidal sinus may be as thin as paper or even absent in places.

The subject of the ocular complications of sphenoidal sinusitis and their cure by operation, as well as the method of operating on the cavernous sinus through the maxillary antrum and nose, are briefly touched upon.

In conclusion, Dr St Clair Thomson urges the necessity of operation in all cases of suppuration in the sphenoidal sinus, and of the examination of these cavities in cases of headache, neuralgia, meningitis, and thrombosis of the ophthalmic vein or cavernous sinus.

J. S. FRASER.

**A CASE OF ACROMEGALY WITH A LESION OF THE HYPO-  
(427) PHYSIS AND OF THE SELLA TURCICA.** (Un cas  
d'acromégalie avec lésion de l'hypophyse et de la selle  
turcique.) GAUSSEL, *Nouv. Icon. de la Salpêtr.*, July-Aug. 1906,  
p. 391.

THE patient was a man aged 65, who came under treatment for Bright's disease and was found on examination to present most of the symptoms of acromegaly. The feet, hands, and lower jaw were enlarged, and there was a kyphosis of the upper dorsal region. The superior maxilla was not hypertrophied and the tongue was of normal size. He died of uræmia of the pulmonary type. On examining the brain it was found that there were numerous meningeal adhesions surrounding the region of the sella turcica. The pituitary body consisted of a thick pulp occupying the sella turcica and also a cavity large enough to admit the tip of the index finger hollowed out in the sphenoid. The pedicle and the anterior and posterior portions of the gland could not be differentiated, and only with difficulty were two small fragments of gland tissue obtained for examination. The pathological process appeared to be one of adenomatous degeneration and cellular infiltration. The thymus and thyroid glands, the brain, cord, and nerves were normal. The case is recorded as further evidence of the etiological relationship between disturbance of the function of the pituitary body and acromegaly.

HENRY J. DUNBAR.

**A RARE CASE OF HYSTERIC TWILIGHT-STATE.** (Ein seltener (428) Fall von hysterischem Dämmerzustande.) WALTHER BAUMANN, *Neurol. Centralbl.*, Sept. 16, 1906, S. 849.

A GREAT number of different pictures, besides the classic form described by Ganser, are grouped under the title hysteric twilight-state. Baumann describes a case, in a tram conductor of thirty-four, who sustained a slight concussion with fractured ribs through a fall from his tram. Three months later, on his return to work, psychical symptoms appeared. These were first evident by his getting lost and being brought home by the police. His state then and on admission was one of extreme excitement and irritability. In his house he brandished a sword violently, throttled his wife after accusing her of infidelity, behaved indecently before his children, and smashed the furniture. He fancied himself in various places and emergencies and had numerous visual hallucinations. He uttered a series of incomprehensible noises, and was completely disoriented as to time and place. The only abnormal physical sign was an increase in the knee-jerks. After two days his speech became occasionally intelligible, but soon relapsed; it was very stammering. There was tremor of the lips, but not of the tongue or hands. On the next day he improved slightly, but intelligence, thought, and attention were greatly deficient. Expansive ideas were marked; he fancied he was the Russian ambassador, etc. He remained in much the same condition for a fortnight, and then a remarkable change occurred coincidentally with his transference to another ward. Practically all his symptoms disappeared in a day, only to be replaced by typical signs of hysteria. These were bilateral concentric visual contraction for all colours, left-sided hypæsthesia to prick, and over a smaller area to touch also, a band on the outer side of the left arm and foot of complete anæsthesia to touch, pain, and temperature; left-sided ageusia. His intelligence was then intact and remained so, as did his speech.

The writer discusses the diagnosis between the condition, which was recognised only after recovery, and G.P.I. and dementia præcox. He excludes a remission of G.P.I. by the completeness and permanence of the recovery in speech and intellect. ERNEST JONES.

**THE RESPONSIBILITY OF HYSTERICS.** (La responsabilité des (429) hystériques.) LEROY, *Rev. Neurolog.*, Aug. 30, 1906, p. 765.

THIS paper formed the opening of a discussion on the subject at the French Neurological Congress held at Lille, August 1906. Leroy first points out that no rigid rules should be established on the subject on

account of the great variation found in the disease. At present, when signs of gross mental change are present, such as anæsthesia, visual contraction, etc., most experts hold for a diminished responsibility, even when the act has no direct connection with the neurosis. Leroy, on the contrary, maintains that such cases should be treated entirely as patients and as having complete irresponsibility. They are cases for an asylum rather than a prison, as medical treatment is needed for the contraction of the social feeling that takes place as much as for the contraction of the visual field. At the same time it may be held that hysterics are responsible for certain acts and not for others, so that both the act and the attendant circumstances must be taken into consideration. Of especial importance are the fugues, thefts, etc., committed during somnambulism, or when the disaggregation has proceeded to such a degree as to produce an automatic or secondary state with evident modifications of character. The most difficult cases to decide about are those showing the different varieties of twilight-states. It must be remembered that, owing to their increased suggestibility, the resistance offered by hysterics to the various impulsions to which they are subject may be so enfeebled that they cannot be regarded as in any way responsible in the sense that the word is used with normal people. In addition to simple hysteria we have often to deal, in a given case, with degeneration, intoxications, and other complications. In conclusion, Leroy strongly maintains that the compromise adopted by most alienists in advocating partial responsibility, a condition which is not recognised by the law, is irrational and absurd, so that we should decide always to treat a patient either as completely responsible or quite irresponsible for his crimes. The feeling of the meeting, particularly as voiced by Grasset and Régis, was in favour of the scientific recognition of partial responsibility, the idea to which the law would sooner or later have to conform, but in the meantime it would be better to treat such cases in the way Leroy suggests, as being pathological and quite irresponsible.

ERNEST JONES.

#### **TRAUMATIC NEURASTHENIA IN THE ARTERIO-SCLEROTIC.**

(430) (*La neurasthénie traumatique chez les artério-scléreux.*) E. RÉGIS (of Bordeaux), *Journ. de Méd. Lég.*, Feb. 1906, No. 1.

RÉGIS insists on the close relationship between neurasthenia in general and arterio-sclerosis, and refers them both to a similar origin, either toxic, infectious, or of the nature of physical or emotional strain. Of twenty cases of traumatic neurosis examined, sixteen were men, and these were over forty years of age, and were

definitely arterio-sclerotic. The neurasthenic state in these cases persisted and tended to become worse. According to the author the trauma frequently merely elicits the symptoms of a latent arterio-sclerosis. In cases of traumatic neurasthenia the expert must pay particular attention to the presence of arterio-sclerosis as determining the prognosis, and as being an important factor in the appearance of the neurosis.

C. MACFIE CAMPBELL.

**TETANY: A REPORT OF NINE CASES.** CAMPBELL P. HOWARD, (431) *Amer. Journ. Med. Sciences*, Feb. 1906.

THE author gives a series of nine cases of tetany supervening respectively upon the following conditions:—

1. Gastric ulcer (?); hyperacidity; hypersecretion; male aged 46
2. Dilatation of stomach; male aged 24.
3. Dilatation of stomach; hyperacidity; male aged 52.
4. Pyloric stenosis; enormous dilatation of stomach; death; *autopsy*—condition probably due to cicatrisation around an old ulcer; male aged 58.
5. Hyperacidity; dilatation of stomach (?); male aged 18.
6. Dilatation of stomach; male aged 45.
7. Chronic diarrhoea; male aged 24.
8. Rickets (?); male infant aged 6 months.
9. Gastro-enteritis; abscess of scalp; rickets; female infant aged 3½ months.

*Summary of Cases.*—Seven were adult males, two were young infants. Four of the adult cases had definite dilatation of the stomach, in one case due to pyloric stenosis. Two of the other cases suffered from hyperacidity of the gastric juice without evident dilatation, making in all six cases of gastric origin. One case suffered from chronic diarrhoea, the only adult case of tetany of purely intestinal origin recorded in America during the last decade.

All the adult cases had definite prodromata such as numbness, tingling, etc., and in all the cases the typical spasms occurred, four being severe, three moderate, and two mild in their intensity.

*Trousseau's phenomenon* was present in five out of six cases examined for it. *Chvostek's sign* was positive in four out of six cases. *Erb's phenomenon* was demonstrated in four out of five cases examined. *Albuminuria* was present in six out of seven cases examined. A detailed analysis of the author's cases is given, followed by a short review of the work of some of the other observers who have studied the subject of tetany.

With regard to the pathogenesis of the disease, the author

favours the modern auto-intoxication theory. He deals with the symptomatology, differential diagnosis, prognosis, and treatment in some detail. He follows most recent authors in advocating lavage in gastric cases, and surgical interference in suitable cases. Thyroid treatment should be tried in cases where the action of the gland appears deficient.

The paper finishes with a tabular analysis of the cases of tetany in adults and in children occurring in America during the last ten years.

W. E. CARNEGIE DICKSON.

**A CASE OF PARALYSIS OF MOVEMENT UPWARDS AND  
(432) DOWNWARDS. (Ein Beitrag zur isolierten Blicklähmung nach  
oben und unten.)** TÖDTER (Hamburg), *Klin. Monatsblätter für  
Augenheilk.*, August 1906.

THERE is a great contrast between the frequency of interference with lateral movements of the globe and the rarity with which the vertical movements are affected. Theoretically a lesion causing this latter manifestation may be situated cortically (or sub-cortically) in the region of the nuclei, in the hypothetical supra-nuclear centre about the corpora quadrigemina, or peripherally. First, the author, in the discussion of the problem, dismisses those cases in which other movements of the eyes are affected, whether this other symptom is or is not synchronous with the vertical defect, as being due to some affection of the third nucleus. Those cases should also be put into a separate category in which there is congenital defect of upward movement along with ptosis, for the situation of the lesion with them is most frequently peripheral—in fact, a muscular anomaly in certain instances, though in others the fault is in the nuclei. The particular affection with which Tödter has to deal is sharply to be differentiated from either of these forms.

So far as a cortical lesion is concerned, we are still quite in the dark; there is no case on record of a paralysis of this nature in which a cortical lesion has been proved post-mortem; but judging from analogy, one cannot but suppose that there is a cortical centre for this movement, though experiments on the lower animals have not proved very convincing as to its precise situation. Clinically, it is true, hysterical paralysis of upward and downward movement has been noted, and the same failure after a head injury with concomitant symptoms pointing to a cerebral situation.

To turn to the hypothetical supra-nuclear centre, several authors place this in the corpora quadrigemina, while others deny its existence, and say it is not necessary to postulate any such mechanism at all. If they are correct, then it is not to be

wondered at that paralysis of these movements occurs so seldom, for the lesion would have to damage the nuclei of the two elevators (or depressors) of each eye. As a rule, it is both upward and downward movement which is paralysed; next most frequently paralysis of upward movement alone; paralysis of downward movement alone has not been recorded.

Clinical observations throw little light on the question of the inter-relations of movements of the globe and the corpora quadrigemina. The symptoms of a lesion affecting the latter are supposed to be cerebral ataxia and paralysis of eye muscles, but even these are not necessarily present, and the case may be indistinguishable from one of cerebellar tumour. When one examines the records of cases of isolated paralysis pursued to post-mortem examination it appears that, with one exception, the cases have all shown a tumour implicating the corpora, or pressure exercised upon its neighbourhood, especially by the pineal gland. In the exceptional case of Thomsen the region seemed entirely unaffected.

Close examination of the post-mortem records shows, however, that much reliance cannot be placed upon the localisation, for it is not possible to exclude pressure effects or "Fernwirkung." Besides that, there are cases also in which there had been no such symptoms, although the corpora quadrigemina were quite destroyed by tumour growth. Nor do the results of experiments on animals do much to clear up the difficulty. But, looking at the matter broadly, one is fairly justified clinically in diagnosing the presence of a lesion about the corpora quadrigemina when one meets with an acquired isolated paralysis of upward or downward movement.

In recorded cases the cause has generally been tumour of one kind or another, much more rarely trauma, hæmorrhage, or "hysteria"; accompanying symptoms have in some instances been conspicuously absent. A difference may be noted according as the lesion is above the association centre actually or in it. If the former, reflex movements of the globe are not interfered with, though the voluntary movements are lost; if the latter, both are alike impossible. Thus in the hysterical cases the patient cannot move his eyes—let us say—downwards, but if one causes him to fix a stationary object and then slowly raises his chin, the eyes will remain fixed upon the object, *i.e.* the reflex movements are not impeded.

Prognosis varies greatly according to the situation of the lesion, but recovery is very rare, to judge from the literature, for Tödter has only found records of three instances. In one of these trauma was the cause, in the other two there was no certainty as to the origin.

Tödter then proceeds to describe two cases which he had seen

at the Breslau clinique. The first was that of a man of 38, who first complained, in March 1900, of double vision and giddiness, which continued even when the eyes were shut. At that date he could not, on desire, move the eyes either up or down, but on making him fix an object and tipping back his head a little degree of descent of the glance took place. Both eyes were equally affected, and on attempt to look up nystagmus came on. Lateral movements and convergence were quite good. Vision was normal, so was the pupil reaction. The urine contained sugar. He practically recovered in a few weeks, and three years later was quite well. It seemed probable that the cause was a small hæmorrhage implicating the fibres coming to the corpora quadrigemina rather than the corpora themselves, since there was the decided difference above indicated between the voluntary movement and the reflex.

The second case was that of a man of 24, who at 17 suffered from a heart lesion. In October 1902 he became suddenly giddy and began to see double, but had neither headache nor vomiting. The left eye stood somewhat higher than the right; downward movement was somewhat nil, even on vigorous effort; upward movement quite impossible; there was double vision, one image standing above the other. The fundus was quite healthy and lateral movements were not impeded. Next day diplopia was gone and all movements were quite free. A year later there was again a little diplopia as before, and limitation of both upward and downward movements. The day following the examination the patient died of pulmonary embolism, and the whole of the nuclear region and of the corpora quadrigemina was examined with great care, but no hæmorrhages or other changes of any kind could be discovered. The only suggestion is that of a soft embolus in one of the vessels of the corpora quadrigemina which had rapidly broken up and been carried away, but it would be strange if this had occurred twice and left no trace discernible on microscopic examination.

Tödter concludes his paper with a brief account of the case of an infant of five months with a high degree of ptosis and with complete inability to turn the globes upwards. The child was in every other respect quite healthy. The interesting point is that the father had an exactly similar defect; further back in the family he did not succeed in tracing it.

W. G. SYM.

#### **PARALYSIS OF THE ABDUCENS IN THE COURSE OF OTITIS.**

(433) TERSON (Toulouse) and A. TERSON (Paris), *Annales d'Oculistique*, July 1906.

THE appearance of ocular paralysis in the course of otitis media is alarming, and highly suggestive of meningitis or sinus thrombosis.



But as an isolated symptom, and even when associated with optic neuritis, its import is not always so serious, and complete recovery may be hoped for. Such a happy issue occurred in cases recorded by Gervais, Keller, Styx, Boerne, Valude, and others, and in the two following under the care of the authors:—

CASE I. was that of a girl, *æt.* 12, with no family or personal history suggestive of syphilis or tubercle. She had had no illness apart from ozæna and two attacks of right otitis media. During the first attack of otitis she had right facial paralysis, which lasted six weeks. Fifteen months later a second attack was ushered in with symptoms of acute mastoiditis, but operative interference was not required in view of the disappearance of symptoms which occurred along with the onset of otorrhœa on the twentieth day. With this, however, right abducent paralysis appeared, though visual acuteness and the appearance of the fundus oculi were normal. Under treatment by mercurial inunctions and iodide of potassium the otitis and paralysis improved concurrently, and were cured three months after the first sign of diplopia.

CASE II. A boy, *æt.* 7, having no stigmata of hereditary syphilis, but whose father was a general paralytic and syphilitic, suffered from left purulent otitis media, probably of influenzal origin, and left abducent paralysis, which came on about a fortnight after the otitis. There was no affection of the fundus or reduction of vision, nor any mastoid symptoms. Treatment consisted in the administration of syrup. iodotannic, and recovery was complete.

The possibility of the paralysis being a mere coincidence is highly improbable, as it always occurs on the same side as the otitis, and may be associated with optic neuritis, and occasionally with paralysis of the third and fourth nerves. The origin of the paralysis may be infectious or reflex. The nuclei of the sixth, seventh, and eighth nerves are closely related functionally and anatomically, and through the carotid plexus the sixth may be reflexly connected with several cranial nerves, and in the walls of the cavernous sinus it receives a direct communication from the ophthalmic division of the fifth nerve, and possibly the third nerve.

Such symptoms as blepharo-spasm, nystagmus, and spasmodic squint, occurring in the course of disease or operative treatment of the ear, are often doubtless of reflex origin; but a paralysis coming on some days after the onset of otitis, and disappearing concurrently with the latter, is much more likely to be of infectious origin. The paths of infection are probably varied. Direct infection would be possible in cases of necrosis or tubercular caries of the petrous temporal at the point where the nerve is in intimate contact with the bone. Infection along the nervous communications of the carotid plexus or localised toxic or necrotic neuritis, at a distance from the seat of the disease, are all very improbable in

the authors' opinion. By far the most likely method of infection is along the venous and lymphatic communications between the carotid canal and its venous plexus and the lymphatic and venous plexus of the tympanum through the carotico-tympanic canals. This communication may become more free owing to the absorption of the bony wall of the carotid canal, thus bringing the carotid sheath and the tympanic mucous membrane into actual contact.

J. JAMESON EVANS.

**A STUDY OF THE PARALYSES OF PNEUMONIA IN ADULTS.**

(434) (*Étude sur les paralysies pneumoniques chez les adultes.*)

PIERRE DAIREAUX, *Arch. Gén. de Méd.*, Sept. 4, 1906, p. 2241.

THE paralyses associated with pneumonia in old people are invariably hemiplegic, nearly always fatal, and frequently found to be due to hæmorrhage, softening, or local changes, the result of atheroma. In the adult, on the contrary, various types of paralysis are found, and recovery can usually be counted on, no matter how grave the symptoms may appear at the time of onset. During the acute stage of the disease, hemiplegias alone are encountered, occurring usually a few days after the commencement, but in exceptional cases preceding by some days the first appearance of symptoms of pneumonia, or even not developing till after the crisis. A right-sided hemiplegia is the more common and is often associated with aphasia. In some cases hemianæsthesia has been noted. Although admitting that in many respects the symptoms in these cases suggest a hysterical origin, the author is of opinion that they are due to a temporary affection of the meninges over the Rolandic area, caused by pneumococcal toxins and comparable to the meningitis known to complicate such diseases as scarlet fever, typhoid, and mumps.

Following the subsidence of the pneumonia a typical peripheral neuritis is occasionally noted, the arm on the same side as the affected lung being usually attacked. These also the writer ascribes to toxic infection, and not, as has been supposed, to a direct extension of an inflammatory process from the pleura. During convalescence, usually some weeks after the attack of pneumonia, generalised paralyses occur, resembling those following diphtheria. There is weakness of the legs and arms, and sometimes involvement of the cranial nerves. The majority of such cases are undoubtedly due to a polyneuritis, but exceptionally, as in a case fully recorded in the paper, the symptoms point rather to a poliomyelitis. With reference to the etiology of the above conditions the writer emphasises the fact that pneumonia is a

general disease and that the paralyses are therefore probably of toxic origin and similar to those associated with the infectious fevers.

HENRY J. DUNBAR

**JACKSONIAN AND PSEUDO-JACKSONIAN EPILEPSY.** Vor-  
(435) KASTNER, *Berlin. Klin. Gesell.*, Oct. 9, 1905, xlii., 1321.

A PATIENT was brought to the Nerve Clinic with a history that convulsive spasms had appeared suddenly in the left arm, extending to the left side of the face and the left leg, and leaving a sensation of weakness on that side. The patient had acquired soft chancre in the year 1897, but had previously been healthy. She stated that three months previously the illness had commenced with irritation in the left arm, and the hand became weak. Later a small swelling was removed from the forehead, and, subsequently, enlargement of the glands on the left side of the neck and in the axillary region was noticed, and these were also removed. On admission the spasms occurred frequently. They commenced by clonic convulsions of the flexors of the thumb and fingers of the left hand, rapidly extending to the left lower facial muscles, and finally to the ocular muscles. Sometimes the spasm first appeared in the elbow and extended to the shoulder. On examining the patient there was found to be definite paresis of the left lower facial muscles. There was also marked paresis of the left arm and hand, especially the flexors of the fingers, those muscles which had been most severely affected by spasm suffering most from paralysis. The left leg was somewhat weaker than the right, and the extensors of the fingers, which at first functionated well, became weak. The fundus oculi appeared to be normal. The glands, which had been removed from the neck and axilla, proved to be malignant and of an endothelial character. It seemed probable that the cerebral symptoms were due to a tumour, the position of which could be ascertained with a moderate degree of certainty. It was thought that possibly it had not developed strictly in the cortex, but just beneath it. The question naturally arose as to whether removal of the tumour should be attempted, but this was abandoned on account of the cachectic condition of the patient.

DONALD ARMOUR

**NOTE ON A RARE FORM OF PSEUDO-ÆSTHESIA.** (Observa-  
(436) tion d'une forme rare de pseudoesthésie.) MATTIROLLO, *Journ. de Neurol.*, Aug. 5, 1906, p. 281.

PSEUDO-ÆSTHESIA is a false but physiological mental perception of colour, sound, smell, taste, or touch, having a real objective origin

from its own or from one of the other four senses, or being of purely psychic derivation. Usually only one such anomaly is present in one patient, but two or more may co-exist. Frequently there is a reciprocity between two senses, a primary sensation of one producing always a secondary false perception of the other, both being indifferently primary or secondary. The condition cannot be regarded as really pathological, as it exists in normal individuals and is always found to have been present since early life. The case recorded is that of a man aged 30 years, a little nervous and excitable, but perfectly healthy and highly intelligent. Since childhood certain words had the effect of producing in him certain gustatory sensations—a state of matters which he had only gradually realised to be peculiar to himself. He was an Italian, but had learned French at an early age, and some years later had acquired a knowledge of German. French and Italian words produced the false perception equally well, but their German equivalents had no effect. It mattered not whether he heard the words spoken, himself uttered them aloud, or merely thought them. Certain words always evoked particular taste sensations, and when eating any pleasing article of diet he automatically pronounced in his mind the corresponding word, which latter proceeding gave him a more complete appreciation of the taste of what he was eating. The word *Russe* called up the taste sensation of pears; *admettre*, of tomato sauce; *Mercredi*, of gooseberry ice, etc. Mere elementary taste perceptions, such as bitter and sweet, were never produced. The patient's other senses were unaffected. The writer would regard the condition as being due either to an association of ideas originating in childhood and developing unconsciously, or to a special psychical process of the nature of a hallucination.

HENRY J. DUNBAR.

**ZUR RECURRENTENFRAGE.** GRABOWER, *Arch. f. Laryngol.*, Bd. 18, (437) H. 3.

ALTHOUGH the vocal cords assume finally the cadaveric position after section of the recurrent laryngeal nerve, in a preceding stage they occupy the median position. This is due to the action of the cricothyroid muscle. The following case demonstrates the accuracy of this view. In a patient whose right recurrent was divided during an operation for thyroid enlargement, shortness of breath supervened shortly after the operation although the voice remained clear. After four days the voice became quite hoarse and the dyspnoea disappeared. The patient was examined a few days later, when a right-sided recurrent paralysis was found.

With reference to Semon's teaching that the abductor muscle

is the first to be affected, the author believes that this is due to this muscle having a smaller mass than the adductors. He says that clinical experience and experiment confirm this view. In lesions of the nerve trunk, whether due to a central or peripheral cause, the nerve elements for both abductors and adductors are equally affected, but the result is first seen in the case of the adductors. The author has shown, by making serial sections of all the intrinsic muscles of the larynx, that the number of the nerve fibres is relatively and actually fewer in the abductors than in the adductors, and in this he finds the explanation of the lesser resistance of the former.

W. G. PORTER.

**A CASE OF "STAMMERING GAIT." (Ein Fall von "Gehstottern.")**  
(438) E. TRÖMNER, *Neurolog. Centralbl.*, Sept. 16, 1906, S. 857.

THE author has in a previous work maintained that stammering is really a Zwangsneurose of the speech apparatus, the reasons being the absence of organic findings, the disappearance of the symptom when the patient is alone, the fact that the stammering is more marked in ordinary speech than in foreign speaking or in singing or whispering, and the influence on the affection of the person's conception of his environment. The tics, as defined by Brissaud and Meige, and hysteric abasia, belong to this same group of Zwangkinesias. Closer analogies with stammering are found in the Harnstottern or begaïement urinaire, in which the individual cannot pass water in the presence of another; but no cases are on record of a pure stammering motor affection of the limbs. Bruns published a case of hysteria in a boy with this as a symptom of the condition. Trömner describes an interesting case of a man, aged twenty-three, who, for eleven years, had suffered from attacks of the following nature. On attempting to rise from a sitting posture, or to start moving from a resting upright posture, he was seized with a cramp which, beginning in the leg, often spread over the whole body; these were nearly always on the right side. After a powerful effort he was enabled to raise his leg, and, once started, went quite well. The attack was accompanied by a psychical condition akin to that found in certain cases of obsessive states; the feeling of distress and fear of not being able to move was in bad attacks extreme. It was assuaged by turning his head to the right, stretching his body backwards, and working his arm violently in the air; he could thus master the difficulty, and relief at once followed. Of late years the attacks had increased in frequency and occurred every day. They were always worse when some one else was present. A short course of hypnotism treatment was very successful, and for six months the patient has been nearly well.

ERNEST JONES.

**A LECTURE ON HEAD-NODDING WITH NYSTAGMUS IN (439) INFANCY.** GEORGE F. STILL, *Lancet*, July 28, 1906.

DR STILL commences by considering the three main symptoms of the condition—the head-nodding, the tendency to look out of the corner of the eyes, and the nystagmus. Of 31 cases which had come under the author's observation, the variety of the rhythmic movement of the head was noted in 22. Eleven patients showed only lateral rotation; six showed only antero-posterior nodding; four either of these movements at different times; and one the combined pendulum movement.

Emphasis is laid on the fact, as being of importance in diagnosis, that the head movement occurs only when the child is sitting with the head unsupported, and that it ceases therefore when the child is lying in a cot or leaning the head against the mother's arm.

An absent-minded stare is frequent in these children, but there is no ground whatever for regarding this peculiarity as due to petit mal, and the author has not seen the least tendency to epilepsy of any sort.

The habit these infants have of looking out of the corner of the eyes while the face is turned in the opposite direction, and slightly downwards, is very remarkable and very characteristic.

The nystagmus is characteristic and peculiar in certain respects, and chiefly in two points. (1) Its unilateral predominance. (2) Its onset without apparent cause in an infant a few months old, and its complete disappearance after a few weeks or months.

As regards the ætiology of the condition, rickets is present in a large proportion of the cases, but rickets is not an essential factor in its causation.

The author agrees with Henoch that the irritation of teeth is the usual exciting cause of the condition :—

“ Its occurrence in rickety children and after the exhaustion of various illnesses or the shock of an injury, and its very close relation to dentition and the exacerbations just when a tooth is in process of eruption, all suggest that spasmus nutans is a functional disorder depending upon an acquired or congenital nervous instability, with some peripheral irritation as an exciting cause.”

Dimness of light has recently been put forward as a cause of the condition, and the arguments adduced in favour of this theory are :—

1. The fact that infants with spasmus nutans often live in dark dwellings.

2. The rarity or non-existence of the disorder amongst the wealthier and consequently better housed.

3. The onset of the disease nearly always during the dark months of the year.

4. The analogy with miners' nystagmus, with which there are known to be associated in rare instances some rhythmical swaying movements of trunk and head.

The difficulties in accepting this theory are :—

(1) It is quite certain that spasmodic nutans may occur in infants living in well-lighted dwellings.

(2) The disorder is undoubtedly seen chiefly in hospital practice, but the author has seen it in three cases in private practice amongst people in comfortable circumstances and well housed.

(3) The seasonal incidence of the disease is indeed remarkable, but a consideration of other conditions leads the author to conclude that during the season November to March, there is some influence, apart from deficiency of light, which favours the onset of certain nervous disorders in connection with rickets.

(4) The analogy with miners' nystagmus does not support strongly the defective light theory.

As regards prognosis, the author states that "it is probably safe to give an unqualified good prognosis," while treatment should be conducted along general lines, benefit in many cases being obtained also from sedative drugs. A. DINGWALL-FORDYCE.

## PSYCHIATRY.

### ON THE CLINICAL SYMPTOMS, DIAGNOSIS, AND PROGNOSIS

(440) OF AMENTIA. (Zur Klinik, Diagnose und Prognose der Amentia.) W. STROHMAYER (of Jena), *Monatsschr. f. Psych. u. Neur.*, May, June 1906.

AFTER a short historical sketch the author analyses the cases of acute hallucinatory confusion received in the clinic at Jena from 1885 to 1902. He starts with a broader group than the amentia of Kraepelin who, by separating the infection psychoses and collapse delirium, and by referring other cases to the manic-depressive or dementia præcox group, has limited the number of cases regarded as true amentia.

Of the 3000 male admissions in the period used, 40 were cases of amentia; among 2500 females, there were 90 cases. Of the 130 cases, only 110 could be used, as the others could not be followed for a satisfactory length of time; in 110 cases the subsequent outcome of the case was ascertained.

Thirty-three cases were of puerperal origin; 40 showed some

marked ætiological factor of somatic nature; in 26 no somatic cause was made out, but emotional strain played an important rôle. In 90 per cent. of the cases it was possible to find some cause responsible for the onset of the disorder; in the total absence of any exogenous factor the diagnosis of amentia should be made with care.

Strohmayer gives a clinical picture of the disorder as presented by his cases. The onset is sometimes abrupt, sometimes preceded by a variety of subjective disorders; the clinical picture quickly develops and presents essentially a state of confusion with prominent hallucinations, transitory delusional elaborations, and general excitement. When the excitement subsides somewhat the fundamental disorder comes out more clearly, viz. the confusion and primary incoherence of thought. This incoherence is the most fundamental and most persistent feature, being well marked even in the remissions and during convalescence; it may be complicated by a secondary incoherence due to the influence of the crowd of hallucinations.

The mood is seldom uniform, but varies quickly and in accord with the trend of ideas. In 10 cases anxiety predominated throughout the whole course; these patients were men in advanced life. Hallucinations and illusions are prominent and lead to a complete misinterpretation of the whole situation. The author had no case similar to that of Weber, where the hallucinatory element was absent, and the essential features were primary incoherence, disorientation, perplexity, variability of mood, and delusions.

Where a secondary incoherence due to the prominent hallucinations complicates the condition, dream-like states with complete disorientation result; this form may be described as acute hallucinatory confusion, while the simpler picture may be considered simple amentia. Clinical records are given to illustrate these types.

Agitation is a well-marked feature of amentia, and may pass into frenzied excitement, and this is not infrequent in certain fatal cases, with emaciation and fever. Agitation may be replaced by stupor, or the two may alternate. The symptom-complex is complicated in various ways by the introduction of so-called catatonic symptoms — verbigeration, mannerisms, stereotypies, mutism, bizarre attitudes. Stereotypies and negativism were in the cases of the author more frequent than *flexibilitas cerea*, echolalia, or echopraxia. In 50 per cent. of the cases some of these symptoms were present, and their significance is discussed later.

The course of the disease is remittent, the incoherence being marked even in the remissions; on recovery, memory of the attack is poor with many gaps, but it is much better for conditions of



hallucinatory stupor than for incoherent and for true catatonic phases.

Of the 110 cases, 21 died in the hospital, 66 were discharged recovered or improved, 23 passed into chronic conditions or had recurrent attacks of a periodic psychosis. The large mortality was due to the accompanying bodily diseases. Of the cases discharged, 61 were followed: 55 remained healthy and showed no defect; while of the other six, one developed general paralysis, three died from a subsequent attack of delirium acutum, two had another attack of amentia.

The records of the 23 unfavourable cases are briefly given; the large majority turned out to be cases of dementia præcox, and this group included only young patients. In other cases the secondary dementia was not of the dementia præcox type. A third group belonged to the periodic psychoses. Where the patient seemed to recover and later had an attack leading to the dementia præcox deterioration, the remission seldom lasted more than three years; the lapse of this period without ominous symptoms justifies the belief that the recovery is permanent.

The prognosis of amentia is very favourable, but one has to consider that in young people an attack of hallucinatory confusion may be the beginning of dementia præcox. Cases with no adequate ætiological factor are specially to be suspected. It is difficult to differentiate between amentia and the delirious form of manic-depressive insanity, and therefore the possibility of a second attack of mental disorder must be kept in mind. The greatest difficulty lies in the separation of certain cases of hallucinatory confusion from Kraepelin's dementia præcox. In dementia præcox the disorder develops more slowly; in the remissions the catatonics are oriented and alert, and there is not that incoherence which is so characteristic of amentia. So-called catatonic symptoms occur in both disorders, and do not make the prognosis of amentia graver. An unfavourable outcome may sometimes be anticipated, not owing to the presence of catatonic symptoms, but on account of the want of harmony between the affective and the intellectual functions.

The disorder is not common, and formed less than 2 per cent. of the admissions.

C. MACFIE CAMPBELL.

**ON HYSTEROMELANCHOLIA.** (Ueber Hysteromelancholie.) G. (441) SPECHT (of Erlangen), *Centralbl. f. Nervenh. u. Psych.*, July 15, 1906.

SPECHT maintains the existence of a special form of melancholia which is based upon the hysterical constitution; he does not use

the term hysteromelancholia to denote the appearance of another psychosis in a hysterical patient, or to characterise the presence of some hysterical features in the course of a melancholia. He does not give any clear delineation of the psychosis, but discusses it in general terms, and criticises the attitude of various authors. It is wrong to assume that because the patient is exaggerated in gesture and expression, therefore she is merely coquetting with grief; the depression is real and often profound.

The occasion for the onset of the psychosis is not necessarily an acute shock; chronic worry is more important. Where the cause is a lively emotion, the latter may be joy as well as grief.

Threats of suicide are frequent and prominent, and cannot be ignored, as the patient may carry out her threat. The psychosis is as a rule polymorph and variable, but on the other hand it may show little variation.

Hysterical stigmata may disappear during the psychosis. The delusions are no more apt to take on religious sexual colouring than in other psychoses.

C. MACFIE CAMPBELL.

#### ON THE SYMPTOM-COMPLEX OF PRIMARY INCOHERENCE

(442) **WITH EXCITEMENT.** (*Ueber den Symptomenkomplex der primären Inkohärenz mit Erregung.*) R. FELS (of Jena), *Monatsschr. f. Psych. u. Neurol.*, May 1906.

FELS reports the cases of three patients presenting incoherence with excitement, without any of the characteristic symptoms which would warrant one in associating the cases either with the manic-depressive, dementia præcox, or amentia group. The incoherence was primary and not secondary to hallucinations, memory defect, or flight of ideas. The mood showed no constant anomaly, but was continually varying, and there were long periods of incoherence without either elation or depression. On this ground, the diagnosis of manic-depressive seemed unjustifiable; the cases were not similar in development to the cases described by Wernicke as confused mania (*verworrene Manie*). Although presenting some similarity to cases of amentia, the well-preserved orientation was in marked contrast with the disorientation of amentia. Even in the presence of some catatonic symptoms the diagnosis of dementia præcox was excluded on account of the absence of defect symptoms and of the age of the patients—40, 34, 46.

With regard to the ætiology, all had bad heredity, and two were of psychopathic constitution; in the third case rheumatic

pains had preceded the onset. In all three cases the clinical picture had already lasted for more than a year.

C. MACFIE CAMPBELL.

**A CASE OF HALLUCINATIONS OF PERIPHERAL ORIGIN.**

(443) (Ein Fall von peripherentstandener Sinnestäuschung.) C. HUDOVERNIG (of Budapest), *Centralbl. f. Nervenh. u. Psych.*, April 1, 1906.

CASE of a neuropathic young man of 18, who for some time had heard sounds, a continuous murmur, an occasional human voice which spoke out his thoughts; this latter phenomenon caused patient great distress. There was no history of ear disease. Examination showed a large amount of wax in the left auditory canal, and a plug of cotton-wool tightly pressed against the drum. The symptoms disappeared completely on removal of the wool and wax. There is no mention of whether the hallucinations were one-sided or not.

C. MACFIE CAMPBELL.

**THE GREAT PSYCHICAL IMPORTANCE OF EAR DISEASE.**

(444) N. S. BRYANT (of New York), *Journ. of Ment. and Nerv. Dis.*, Sept. 1906.

THE author's attention was drawn to this subject by patients who suffered from otitis media with tinnitus, reacted to auditory hallucinations, were treated for the middle ear disease, and the hallucinations disappeared. He reports such cases with recovery and refers briefly to several others. The dry forms of otitis media are the most frequent cause of tinnitus, and are more commonly associated with psychoses than are the purulent forms. Tinnitus from the psychic standpoint may be classified as follows: (1) that not heeded by patient (largest class); (2) that caused by hypochondria, neurasthenia, melancholia, etc.; (3) that causing auditory hallucinations. The hallucinations may be (a) of slight importance, the patient being conscious of them; (b) "unconscious hallucinations" of no great psychic importance; (c) "true delusions" finally becoming organised. Köppe in 1867 examined 100 insane patients with the following results: without symptoms pointing to the ear, 20; auditory hallucinations, 77; marked ear disease, 31; tinnitus aurium, 26. Redlich and Kaufman in 1896 found in the examination of 97 insane patients, 29 doubtful, 10 without auditory hallucinations, 11 normal, 58 with auditory hallucination, 57 with

abnormal ears, 26 with tinnitus. Among their cases hallucinations were most common in paranoia (50) and alcoholic insanity (17).

Bryant examined 36 insane with the following results: 10 doubtful, 4 normal cases, 42 abnormal ears, 5 without hallucinations, 41 with hallucinations, 27 with tinnitus (not classified according to disease).

In regard to prognosis, the author believes the prognosis to be bad for recovery from auditory hallucinations in proportion to the deafness; in old age the prognosis is particularly bad. Unilateral tinnitus does not have so much psychic influence as bilateral.

In conclusion, the evidence points to some connection between ear disease and auditory hallucinations other than mere coincidence; it is probable that auditory hallucinations in most cases originate in subjective ear sensations; cure of the ear disease assists in the convalescence from the psychosis in many cases. Some cases of insanity appear to be excited by ear disease and convalescence is delayed by its presence; unilateral hallucinations are unquestionably due to unilateral ear disease.

C. H. HOLMES.

**ON HYDROTHERAPY IN MENTAL DISORDERS.** (Zur Hydro-  
(445) therapie bei Geisteskranken.) W. ALTER (of Lindenhaus),  
*Centralbl. f. Nervenh. u. Psych.*, May 15, 1906.

ALTER answers some criticism by Sanger, who had deplored the limited use of hydrotherapy in the treatment of the insane, and recommended a more complicated hydrotherapy than is usually employed. Complicated hydrotherapeutic treatment is out of place in a large hospital for the insane; the treatment must be left to a large extent to the nurses, and the directions to be given need to be simple and easily understood.

Hospitals for the insane have usually limited equipment, an inadequate medical staff, and an insufficient nursing staff.

Alter looks upon the treatment of the excited cases as the special field for hydrotherapy; rest during the day and sleep at night are what is aimed at. Complicated packing and any method which provokes the resistance of the patient and general antagonism in the ward should be if possible avoided. The packing of a patient in wet sheets until a reaction is obtained, and then repeating the treatment, provokes such antagonism and induces resistance in the patient which leads to dangerous exhaustion.

Warm packs are recommended; vasomotor variation is hindered by the equable moist temperature, and peripheral irritation being removed, the patient is in the most favourable condition for the

inducement of psycho-motor calm. The pack in this respect is more effective than the warm bath. C. MACFIE CAMPBELL.

### TREATMENT.

**ON THE TECHNIQUE OF OPERATIONS ON THE CENTRAL (446) NERVOUS SYSTEM.** Sir VICTOR HORSLEY, *Brit. Med. Journ.*, Aug. 25, 1906.

SIR VICTOR HORSLEY based his Address in Surgery upon an analysis of his cases at the National Hospital, Queen Square, since 1886. He points out that the advance in technique of the surgical treatment of diseases of the brain and the spinal cord has been relatively less than the improvement in our knowledge of the seat and nature of the diseases for which surgical intervention is useful and necessary. And while correct diagnosis in diseases of the nervous system is still far to seek, yet, he adds, operative treatment in such a difficult field is often expected to yield as good results as relatively easier and simpler work.

He lays stress upon the immense importance to the community that the study of neurology should be pushed forward by every means in our power, in order that the earliest commencement of a tumour of the brain should be determined as certainly as that of one nearer the surface of the body. He then briefly alludes to the responsibility of the surgeon in the treatment of diseases of the central nervous system, and complains that outside of the Queen Square Hospital surgical treatment is almost universally regarded as a *dernier ressort*.

Horsley then proceeds to discuss surgical treatment under two aspects, viz.: 1. Palliative Surgical Procedures; 2. Curative Surgical Procedures. He points out that the prominent characteristic symptoms of intracranial disease, viz. (1) optic neuritis, which usually ends in total blindness; (2) severe headache and vomiting, all of which are dependent upon pressure, can be completely palliated or wholly removed by making a sufficiently free opening in the skull and dura mater. Rarely will the opening of the skull alone suffice. This opening should be made preferably in the basal temporal region of the right side. Should the tumour, however, directly involve the optic tract, the specially delicate anatomical structure of the optic tract may prevent the attaining of this result. In referring to the localising value of the incidence of the optic neuritis, Horsley lays down the rule, drawn from an examination of his own cases of intracranial tumour, that the optic neuritis commences on the side of the lesion. While admitting some true exceptions to this rule, he argues that some of the exceptions hitherto described have not been real, for in any

given case it is not a question merely of the number of dioptries of swelling of the disc, but also a matter of the anatomical changes in the disc, and finally, when first seen the disc on the side of the lesion may be actually subsiding into decadent conditions at a time when the opposite disc is rising to its maximal swelling.

Passing to curative surgical procedures, Horsley says it is necessary to consider (1) what is the nature of the disease; (2) what loss or aberration of nerve function it causes; (3) whether, if the lesion be wholly extirpated, there will be a recovery from the disorder of function; and (4) whether any loss which may have been present before operation will be made permanent by the necessary extirpation of particular regions of the brain.

On points like the last he says a satisfactory opinion cannot be given until we learn from the cerebral physiologist under what circumstances and to what extent we can get *compensation* of function when various parts of the cerebrum and cerebellum are destroyed. As regards the cerebrum, from clinical records, Horsley states that special motor functions, probably the special senses, and certainly the hemianopic representation of sight, cannot be restored if the whole of their cortical representation be removed. The higher sensory representations and the intellectual functions are not permanently abrogated by the destruction of any one part of the cerebral hemisphere.

As regards the cerebellum, Horsley is distinctly opposed to the proposed removal of any part of it for the purpose of reaching a deep-seated tumour. He advocates the displacement of the cerebellum, which necessarily involves bruising it, and instances remarkable recoveries from this bruising. Whether the recovery is due to restoration of function of the bruised portions or compensation from the uninjured part, Horsley is unable to say with certainty, though he believes the former. He therefore concludes that as much as possible of every portion of the encephalon which is not absolutely shown to be diseased should be preserved.

Horsley then proceeds to discuss the question of anæsthesia in operations on the central nervous system, limiting his observations to ether and chloroform, as he had never employed the intraspinal injection of cocaine or stovaine, and has entirely given up the combined anæsthesia of morphine with chloroform. He regards ether as inadmissible as an anæsthetic in operations on the central nervous system, because it directly causes, besides a rise of the blood pressure, a notable increase of the blood venosity, and therefore much additional and troublesome hæmorrhage. Chloroform, *per contra*, causes a fall of blood pressure with relatively less blood venosity. It therefore does not aggravate the bleeding, nor embarrass the respiration. It is also attended by

practically no after-excitement. Horsley agrees that chloroform is more dangerous, and that it kills by paralysis of the respiratory centre, as often or more often than by paralysis of the heart. It is especially in cases of increased intracranial tension that the danger of respiratory paralysis is greatest. Therefore, he states, the immediate problem is how to regulate the dosage of chloroform.

Horsley advocates the use of an apparatus for giving known percentages of chloroform, Vernon Harcourt's being the one he has entirely worked with. It is used as follows: Commencing with the dose at 0.5 per cent., and rising in one or two minutes to 2 per cent., the patient is ready for operation in five to eight minutes. The amount of 2 per cent. is given for about five minutes before the skin incision and reflection of the flap which constitutes the maximal pain period of the operation. The bone is removed at 1 per cent. The dura being a sensitive membrane, the dose is somewhat raised just previous to its incision. As soon as the dura is opened the encephalon is dealt with under less than 0.5 per cent. of chloroform in the air respired. The percentage is raised again to 0.7, or even 1 per cent., for the skin suturing.

Passing to the depressant physiological effects of the general anæsthetic, Horsley says all operating-rooms should be kept at a temperature of not less than 75° F., and the operating-table heated. But, in addition, he is strongly of the opinion that to maintain the physiological energy of the central nervous system and prevent shock thereto, it is necessary during all operative procedures on the skull and its cavity to prevent cooling by radiation from the brain exposed in the wound. For this purpose the wound should be constantly irrigated with a solution of 1 in 10,000 sublimate, or with saline, at a temperature of 115°. An additional use of this hot irrigation is the arrest of capillary and arterial hæmorrhage.

Turning to the subject of hæmorrhage, Horsley says that the first general principle is the recognition of the fact that as few vessels as possible should be obstructed, this applying as closely to veins as to arteries. He is opposed to the practice of tying such main arteries as the carotid to control hæmorrhage, owing to serious and even fatal secondary œdema and softening resulting therefrom. He thinks nothing is better than the plan of tying all the arteries around the lesion before extirpating it, beginning the incision in the brain below and carrying it upwards and towards the mesial plane.

Horsley points out how remarkably easy it is to arrest capillary and arteriole oozing from the brain by the simple means of hot irrigation. For this purpose the temperature of the fluid should not exceed 115° F., and should certainly not fall below 110° F.

When a lesion is about to be extirpated, and there is reason to

expect considerable oozing, or when the brain is obviously turgid with congestion, Horsley takes advantage of the fact that chloroform causes a marked fall in blood pressure, and has the chloroform percentage raised for a quarter to half a minute to 1 or 2 per cent. By this means a convenient, proportionate, and temporary anæmia is induced. The bleeding from veins and sinuses in bone is controlled by wax plugging; wounds of the sinuses and Pacchionian bodies and venous lakes in the dura mater are closed by a fine lateral suture on a round needle; and the principal veins are ligatured by passing a round needle beneath them. Venous oozing can be rapidly controlled by the inhalation of oxygen. This is done by raising the percentage of oxygen in the anæsthetic atmosphere breathed, by directing a stream of the gas through the air inlet of the Harcourt regulator.

For the lessening of shock, Horsley always does his brain operations in two stages, opening the skull first, and then about five days after opening the dura and removing the lesion. Regarding the influence of the region opened, he finds that if a line be drawn from the frontal eminences to the occipital protuberance, more shock results from operations below that line than from above, and also proceeding backwards from the frontal to the cerebellar pole of the encephalon. Horsley thinks that the risk of an operation for decompression is greater if the opening for the relief of pressure is not made directly over the lesion.

In the treatment of shock, Horsley advocates the use of inhalations of oxygen in embarrassment of the respiratory centre, and above all in depression of that centre in the use of strychnine, which he, however, adds is a circulatory depressant, and therefore not to be given too freely.

He thinks that cardiac stimulation is a clinical error. He relies upon repeated two-hourly nutrient enemata, with a very small dose of atropine if time presses. In cases of peripheral vaso-motor paralysis he finds digitalis useful, but stops it on any acceleration of pulse. He lays stress upon the main principle of operating on the central nervous system which is the avoidance and prevention of all conditions which lead to shock—namely, cooling and mechanical disturbance of the central nervous system.

Concerning sepsis, Horsley points out the proclivity of the central nervous system to invasion by septic micro-organisms and the extremely feeble degree of its resisting power. For this reason he thinks the less drainage employed the better, and that every effort should be made to close the skin wound as early as possible.

Horsley ends his address by giving an analysis of his Queen Square cases which "brings out in very strong relief the fact that where the technique of intracranial operations fails most is the



treatment of malignant disease." He appends the following table of 55 tumour cases:—

Cases.		
Glioma . . . 19	} 23	Recurrence within 2 years, 20.
Sarcoma . . . 4		
Endothelioma . . . 8	{	1 recurrence 3 years later; died of valvular heart disease. 7 alive well, longest 5 years. 2 died within 3 months of tuberculous meningitis. 2 alive well, longest 7 years.
Tuberculous . . . 4		
Gunma . . . . 8		No recurrence.
Fibroma . . . . 4		Ditto.
Cysts . . . . 5		Ditto.
Adenoma . . . {Pituitary}	{ 3	1 recurrence.
Adeno-sarcoma		

He concludes with the following general deductions on the question of the surgical treatment of malignant disease of the encephalon: (1) That operation should be resorted to as early as possible; (2) the tumour should be, if possible, freely exposed and examined and extirpated with surrounding tissue; (3) that if it cannot be removed without undue interference with important or essential structures, there remains some possibility of the tumour undergoing retrogression in a certain number of cases.

DONALD ARMOUR.

#### RESULTS OF OPERATIONS FOR THE REMOVAL OF CEREBRAL (447) TUMOURS. KNAPP, *Boston Med. and Surg. Journ.*, Feb. 1, 1906.

THE author, in 1889, had collected 24 cases of operation on tumours of the brain. In 1891 he added 48 cases to these, making 72 cases, and in 1899 collected 489 cases more. He compares these three groups with the present collection to see what progress has been made in the surgical treatment of brain tumour, as well as to present the general results drawn from the whole collection of 828 cases. The accompanying tables (Tables I. and II.) show the results of these operations as reported in 1899, the results in 267 cases collected since, and the total results in 828 cases. The subsequent tables (Tables III. IV. V. and VI.) show the mortality, the number of cases not benefited by operation, and the number of cases in which there was a failure to remove the tumour in each of the four groups of cases, together with the total results in the 828 cases. The percentages are given, but he confesses that they are somewhat fallacious. A single case in 1889, when only 24 cases were recorded, accounted for about 4 per cent., but in 1899 a single

case accounted for about one-fifth of 1 per cent. The tables show that in the later cases there has been a diminished mortality as a result of the operation, and a somewhat greater number of cases benefited, but Knapp regards this as being due to the improved surgical technique. He points out that the table showing the percentage of failures to remove the growth is less flattering to the neurologist than the previous table to the surgeon, in spite of the number of cases said to have "recovered." A study of the recorded cases confirms him in the belief that most of these simply lingered on for a time, paralytic, epileptic, or blind, and that in many instances the growth recurred; that the cases of actual recovery, that is, of complete restoration to health, are exceedingly few. He thinks that it is justifiable to operate in suitable cases with the probability of affording temporary relief, but the possibility of a complete cure is slight; and the optimistic views, or rather hopes, so often expressed as to the benefit of operation are hardly justifiable. He thinks that the claim of the so-called palliative operations to relieve pressure can be admitted only in about half of the cases, in which they relieve headache and arrest the progress of optic neuritis.

TABLE I.

1899.	Recovered.	Improved.	Not improved.	Died.	Not stated.	Total.
Removed .	76	72	59	86	18	311
Not found .	4	14	46	61	5	130
Impossible .	0	6	8	29	2	45
Palliative .	0	37	22	12	4	75
Total .	80	129	135	188	29	561
1905.						
Removed .	36	50	19	37	18	160
Not found .	0	10	15	26	8	59
Impossible .	0	5	3	11	0	19
Palliative .	0	11	14	3	1	29
Total .	36	76	51	77	27	267
TOTAL.						
Removed .	112	122	78	123	36	471
Not found .	4	24	61	87	13	189
Impossible .	0	11	11	40	2	64
Palliative .	0	48	36	15	5	104
Total .	116	205	186	265	56	828

TABLE II.

1899.	Recovered.	Improved.	Not improved.	Died.	Not stated.	Total.
Frontal . .	5	2	0	2	0	9
Central . .	43	57	38	52	2	192
Parietal . .	2	3	3	2	0	10
Temporal . .	0	0	3	4	1	8
Occipital . .	1	1	1	2	1	6
Cerebellum . .	1	6	3	8	0	18
Not stated . .	24	3	11	16	14	68
Total . .	76	72	59	86	18	311

1905.						
Frontal . .	10	4	3	6	2	25
Central . .	9	25	5	2	3	44
Parietal . .	5	2	2	10	0	19
Temporal . .	2	4	3	1	0	10
Occipital . .	2	2	1	0	0	5
Cerebellum . .	6	10	5	15	1	37
Not stated . .	2	3	0	3	12	20
Total . .	36	50	19	37	18	160

## TOTAL.

Frontal . .	15	6	3	8	2	34
Central . .	52	82	43	54	5	236
Parietal . .	7	5	5	12	0	29
Temporal . .	2	4	6	5	1	18
Occipital . .	3	3	2	2	1	11
Cerebellum . .	7	16	8	23	1	55
Not stated . .	26	6	11	19	26	88
Total . .	112	122	78	123	36	471

TABLE III.  
PERCENTAGES OF OPERATIVE MORTALITY.

	1889.			1891.			1899.			1905.			Total.		
	Cases.	Deaths.	Per Cent.	Cases.	Deaths.	Per Cent.	Cases.	Deaths.	Per Cent.	Cases.	Deaths.	Per Cent.	Cases.	Deaths.	Per Cent.
Removed . . .	18	7	39	28	8	29	265	71	27	160	37	23	471	123	26
Not removed . .	6	4	67	20	12	60	224	86	38	107	40	37	357	142	43
Total . . .	24	11	46	48	20	42	489	157	32	267	77	29	928	265	32

TABLE IV.  
PERCENTAGES OF FAILURE TO BENEFIT.

	1889.			1891.			1899.			1905.			Total.		
	Cases.	Not benefited.	Per Cent.	Cases.	Not benefited.	Per Cent.	Cases.	Not benefited.	Per Cent.	Cases.	Not benefited.	Per Cent.	Cases.	Not benefited.	Per Cent.
Removed . . .	18	9	50	28	12	43	265	125	47	160	56	35	471	202	43
Not removed . .	6	5	83	20	14	70	224	158	70	107	72	67	357	249	70
Total . . .	24	14	58	48	26	54	489	283	58	267	128	48	828	451	54

TABLE V.

## PERCENTAGES OF FAILURE TO FIND THE GROWTH.

	1889.	1891.	1899.	1905.	Total.
Removed . . . . .	18	28	265	160	471
Not removed . . . . .	5	10	115	59	189
Per cent. failure . . . .	22	26	30	27	29

TABLE VI.

## PERCENTAGES OF FAILURE TO REMOVE.

	1889.	1891.	1899.	1905.	Total.
Removed . . . . .	18	28	265	160	471
Not removed . . . . .	6	20	224	107	357
Per cent. failure . . . .	25	41	46	40	43

DONALD ARMOUR.

**CEREBRAL DECOMPRESSION; PALLIATIVE OPERATION IN  
(448) THE TREATMENT OF TUMOURS OF THE BRAIN,  
BASED ON THE OBSERVATION OF FOURTEEN CASES.**  
SPILLER and FRAZIER, *University of Penn. Med. Bull.*, September 1906.

THE authors commence their article by a review of the literature, and refer to cases of simple trephining of bone for the relief of symptoms of increased intracranial pressure by Annandale, Lister, Sahli, White, Horsley, Jaboulay, Caton, Paul, MacEwen, Taylor, Keene, Bruns, Bramwell, Kammerer, Wyeth, Sanger, Albert, Schlesinger, Putnam, Schultze, Clarke and Morton, Wiener, Rohmer, von Bergmann, Babinski, Leslie Paton, Risien Russell, Codman, and Cushing. After a careful study of these cases, and of the views of the different authors, Spiller considers that the weight of opinion is decidedly in favour of palliative operations. The choked disc, headache, vertigo, nausea, vomiting, and to some extent the convulsions, are all favourably influenced by this method of treatment. Relief from these distressing symptoms is by no means to be despised, even though the tumour is not removable. The relief from many of these symptoms is often permanent, *i.e.* during the period the patient may continue to live, and as the growth of the tumour is not hastened by the palliative operation, and may be slow, we should be thankful for a means of relieving the distressing symptoms of intense intracranial pressure.

Spiller himself is somewhat sceptical as regards the disappearance of the Jacksonian convulsions after merely opening the skull and dura. If the convulsions are very frequent, he is doubtful as to whether palliative operations can cure focal symptoms. Bruns thinks that they cannot do this, but the subject is one that has important bearings. It is probable that a small tumour will cause more local disturbance if the general intracranial pressure is increased, not only because the local disturbance is added to the general pressure, but also because any one part of the brain is more irritable when the disturbance is general. Spiller thinks, however, that this is not sufficiently important to prevent palliative operation, and advises that this should be done before the general symptoms become very intense, and especially before optic neuritis has developed so far that blindness is likely to result. The apparent unanimity of opinion as regards the effect on choked discs of opening the skull makes the necessity of this operation at an early period very evident.

He thinks it is a mistake to regard palliative operation as a substitute for radical operation. The tumour should be removed whenever this is possible, and palliative measures are to be considered only when the tumour cannot be located, or is too large for removal, or possibly is a glioma. He thinks that no surgeon who has had but little experience in operating on the brain should attempt the removal of a brain tumour, and always insists upon a specialist in brain surgery operating upon his own cases. He considers the experience of Horsley as regards atrophy of tumours as a result of palliative operations to be unique. He himself has never found other similar cases, nor has he ever experienced any such result in his own. He has never seen arrest in the growth of a tumour following the removal of a part, but several times has seen increase of symptoms result, and hence dreads the partial removal of a growth, especially if it is a glioma. It seems to him that the congestion of the tumour and of the surrounding tissue, and the greater space afforded for the growth of the tumour after partial removal, favour rapid growth of the remaining portion. He is convinced that it is better to leave the tumour untouched if only a part can be removed, especially if the growth is a glioma. He thinks that the attempt should never be made to remove a glioma, and yet confesses that there is no way of determining before the operation that the tumour is a glioma. He thinks it is a questionable proceeding to excise the portion of brain protruding through the opening in cases of palliative operation. He says it may be thought that the growth of the tumour is arrested by the operation: this is possibly doubtful. Internal hydrocephalus (meningitis serosa) or some other lesion may cause the symptoms of brain tumour, and relief of intracranial pressure may produce great

modification or disappearance of them. Spiller's views, as a result of his experience and of a study of the literature are: (1) Palliative operations should be performed early in every case in which symptoms of brain tumour are pronounced, and before optic neuritis has advanced far, especially when syphilis is improbable, or antisyphilitic treatment has been employed. (2) Partial removal of a tumour, especially of a glioma, is a questionable procedure. (3) Palliative operation does not, under ordinary circumstances, cause atrophy of brain tumour, and probably does not arrest its growth. On the other hand, it probably does not hasten its growth. (4) Palliative operation is not to take the place of a radical operation when the latter can be performed without great risk to the patient. (5) In some cases the symptoms of brain tumour disappear almost entirely for a long time, or permanently, after a palliative operation. This result is obtained either by relief of intracranial pressure, or by the removal of some lesion (meningitis serosa, etc.) other than brain tumour.

Frazier considers that the very vascular infiltrating sarcomata should be classed with the inoperable tumours, and believes that the expectation of life which remains to the patient would be greater in the majority of cases after palliative operation. On the other hand the fibroma, the somewhat definite fibro-sarcoma, and the gumma of limited dimensions, should be classified as of the operable group. The size of the tumour as revealed at the autopsy has no practical bearing at all on the problem. The size of the tumour at the time when the symptoms first made the diagnosis possible only should be considered. If it were possible to ascertain the point from which the tumour took its origin, we should have some valuable statistics as to the operability of tumours. The further away the tumour is from the cortex, or the nearer it is to the base of the brain, the more it approaches the field of operable growth. He considers that if the statistics are to be of much value to the surgeon, they should be based on the records which are made on the operating table, or at the autopsy if the patient dies as the result of the operation. As with carcinoma of the stomach, so with tumours of the brain, the greater the number of the cases brought to the surgeon in the earliest recognisable stage of the disease, the sooner shall we be able to state with some degree of accuracy in what percentage of cases there is a reasonable hope of being able to perform a radical operation. Frazier divides the cases in which a decompressive operation may be required into two classes: (1) those in which there is reason to believe that the tumour cannot be removed in its entirety; and (2) where the tumour cannot be localised, and yet the possible loss of vision, intense headache, and distressing vomiting almost demand some immediate measure of relief.

He thinks that in palliative operations in the cerebellar region, the usual incision, beginning at the mastoid process, and following the line of the transverse sinus to the occipital protuberance, should be replaced by a vertical incision, beginning a little above the superior curve line, and extending downwards for a distance of three or four inches. The edges of this wound can be retracted sufficiently to afford the space necessary to carry out the subsequent steps of the operation. A much more perfect approximation of the wound may be obtained, as the muscles are split in the direction of their fibres, and not in various angles. If for any reason a bilateral craniectomy seems advisable, the muscle splitting operation may be repeated upon the opposite side. He thinks there is less chance of disturbance arising from the traction on the pons or medulla if an intervening bridge of bone is left. He has, failing to find the tumour, in certain cerebellar cases deliberately removed from a quarter to a third of the cerebellar hemisphere, and attributes the relief of the pressure in part at least to this.

In all their cases the operation has been confined to one side, and it was not found necessary to operate a second time for the removal of the bone on the opposite side. Should this be found necessary, Frazier considers that the operation had better be performed at two sittings, seeing that patients with cerebellar lesions are at the best not very favourable subjects for operative treatment. Taking first those cases in which the tumour cannot be localised, Frazier thinks that there are two points in the technique of the operation about which there may be some difference of opinion, namely, the area of the brain to be uncovered, and the incision of the dura.

The decompression operation consists solely in the removal of a portion of the cranial vault, with or without incision into and removal of a portion of the dura. When there is no guide as to the situation of the tumour, the operator is free to select any one of four areas: the frontal, parietal, occipital, or temporal. As Sanger suggests, it is desirable to give preference to that portion of the skull beneath which may be said to exist a silent area of the brain. This is especially true of the right temporal region, so that, in the absence of any contra-indication, this may be said to be the region of choice.

Turning then to those cases in which the tumour has been definitely localised, Frazier agrees that it is impossible to determine prior to the operation whether the tumour is one suitable for a radical rather than a palliative operation. He thinks that the duration of the disease is a guide neither to the nature nor to the size of the tumour. He thinks that in every instance therefore the operator should begin the operation with the intention of attempting to expose the tumour. He should proceed as for the performance of an osteoplastic operation. The flap should be carefully



mapped out and reflected, and the usual exploratory measures adopted. Failing to find the growth, or finding an inoperable one, the propriety of a decompressive operation is clearly indicated. He must then determine as to whether he will remove the bone of the flap, wherever this may be, or close the flap and remove the bone from the area of choice, namely, over the right temporal lobe. As to the escape of cerebro-spinal fluid, there are certain observers, notably von Bergmann, who believe that the beneficial effect of this operation is largely attributable to the escape of cerebro-spinal fluid. It may possibly be difficult to prove or disprove this idea on theoretical grounds, but in their own experience Spiller and Frazier have not been able to note any radical difference in the results between those cases in which the dura was either left intact, or if incised for exploration, was closed immediately afterwards. They consider that the relief, which extends over a period of months or years, can scarcely be attributed to the escape of a small quantity of cerebro-spinal fluid from a temporary opening in the dura. Many consider that the success of the operation depends upon the establishment of an opening in the dura, as well as in the skull, it being said that the dura is not elastic enough to stretch sufficiently when subjected to pressure. Frazier says that the dura will stretch when the overlying bone is removed sufficiently to afford adequate relief of tension, as has been demonstrated in their cases over and over again. He points out that, whatever else may be said in favour of or against the removal of the dura, it should be borne in mind that if the dura is removed, and any complication arises in the repair of the wound, it is more than likely that a fungus cerebri will develop. He confesses that in the hands of operators experienced in this field of surgery the danger is very slight, but thinks that it is questionable whether the removal of the dura should be resorted to as a universal practice. He considers that if the dura is disturbed at all, the operator should be content with making a crucial or radiating incision, and in many cases it may be advisable to reserve this step of the operation until indication arises for further intervention.

He concludes that the decompression operation offers to the patient relief from the three cardinal symptoms of cerebral tumour, headache, nausea, and vomiting, restores vision, and if the lesion is in the cerebellar fossa, relieves ataxia and vertigo. The opportunity to save or restore the patient's eyesight is one of the strongest arguments in favour of the palliative operation. Without exception the choked discs subsided in every one of their cases. The prospect of preserving the eyesight alone therefore would justify the operation, but no less so the opportunity of relieving headache. Their results with respect to headache have been uniformly good. They append a summary of their fourteen cases.

## SUMMARY OF CASES.

Location of Lesion.	Period since Operation.	Results.
Cerebrum.	1 year (operation was not performed, but relief of pressure occurred spontaneously).	Alive and free from pain.
Cerebellum.	7 years.	Almost complete recovery during 2 or 3 years: present condition unknown.
Cerebellum and Cerebrum.	2 years and 7 months.	Alive and free from pain.
Cerebellum.	2½ years.	Alive and free from pain.
Cerebellum.	2½ years.	Alive and free from pain.
Cerebrum.	1 year and 7 months.	Alive and free from pain.
Cerebellum.	8 months.	Second operation was performed: tumour found and removed.
Cerebellum.	7 months.	Died 7 months after operation.
Cerebellum.	7 months.	Alive and free from pain.
Cerebrum.	2½ months.	Alive and free from pain.
Cerebellum.	2½ months.	Alive and free from pain.
Cerebellum.	2 months.	Radical operation was performed 2 months after palliative operation.
Cerebrum.	—	Alive, but little, if any, relieved.
Cerebellum.	About 3 years.	Improved: death from tumour about 3 years after palliative operation.

DONALD ARMOUR.

**OPERATIVE TREATMENT OF PURULENT MENINGITIS.** (Die (449) operative Behandlung der eitrigen Meningitis.) KÜMMEL, *Arch. f. klin. Chir.*, lxxvii., 1905, 77, 930.

THE author considers that the advances which have been made in the operative treatment of purulent peritonitis, and the fortunate results that have been obtained in that of tuberculous peritonitis, make it evident that we are not very far from being able to obtain, by means of a more active operative procedure than that hitherto employed, amelioration or cure in the case of disease processes of the meninges in which formerly nothing could be hoped for. He refers to the free drainage of the abdominal cavity in cases of

septic peritonitis for the purpose of aiding nature in her curative powers, though it is impossible to remove all the toxic foci, and thereby attain in a disease which otherwise would certainly run a fatal course a percentage of cures which is progressively increasing. In cases of tuberculous peritonitis, the abdominal cavity is simply opened, with or without removal of the ascites, and yet cures are obtained without interference with the true primary disease. Judging from these results, he thinks that attempts should be made to obtain similar ones in intracranial inflammatory affections more frequently than has hitherto been done. He thinks that the small proportion of cases which recover after opening of the skull for tuberculosis meningitis is due to the miserable condition of the patients, though after trephining we see a transitory improvement, due to diminution of the pressure, and shown by a remission of stupor, liberation of the sensory faculties, and remission of pain; and he thinks it worth while to endeavour to obtain these results in the face of an otherwise fatal malady. He thinks that the cured cases of cerebro-spinal meningitis reported by Lenharty, in which cure was effected by lumbar puncture, are in favour of the employment of active therapeutic measures in inflammatory diseases of the cerebral and spinal meninges. He is of the opinion that in epidemic cerebro-spinal meningitis, when lumbar puncture fails, free opening of the skull cap or vertebral column should be performed, as affording a prospect of a beneficial result. He refers to the cases of circumscribed purulent meningitis, usually of otitic origin, or complicating fractures of the skull, which have been benefited by operation. He also refers to those cases of circumscribed meningitis, with manifestations of a general meningitis, which have been cured by more or less thorough surgical interference. He refers to cases reported by Lucca, Witzel, Bertelsmann, and M'Ewan, in which successful results have been obtained. He then reports at length a successful case of diffuse purulent leptomeningitis, extending to the cauda equina, and due to a fracture at the base of the skull, which was cured by operative treatment. The cranial cavity was extensively opened on both sides of the occipital bone, so as eventually to allow of an opening to the vertebral canal. The posterior cranial fossa was drained. He thinks the result obtained in this case is decidedly in favour of the opinion of those who advocate active interference, even in diffuse purulent leptomeningitis. Güssenbaur, at a meeting of the Vienna physicians in January 1902, stated that the treatment of meningitis belonged to surgical therapeutics of the future. Kümmel thinks that the fact that the possibility of attaining a cure does present itself, even in severe cases, in which otherwise there is no room for hope, through extensive opening of the cranium, must incite us to interfere surgically in the earliest

possible stages of inflammatory diseases of the meninges, and thus endeavour to aid nature in her attempts at cure.

He concludes that the opening must be made as early as possible, either in the cranial cavity or the vertebral canal, or even in both, after meningitic symptoms appear, if we are to save an otherwise doomed patient.

DONALD ARMOUR.

#### **ON FACIAL NEURALGIA AND ITS CURATIVE TREATMENT**

(450) **BY EXCISION OF THE GASSERIAN GANGLION.** JORDAN-LLOYD, *Birm. Med. Review*, Jan. 1906.

THE writer regards instantaneous relief of the agonies of inveterate epileptiform neuralgia, by a carefully-planned and well-executed excision of the Gasserian ganglion, as one of the most satisfactory operations in the whole field of surgical work. He regards the operation as having passed its period of probation, and thinks that the remarkable relief the operation brings at once to the almost distracted victims of this cruel disorder should make us confidently advise our patients to accept it. He is careful to distinguish between the many varieties of facial neuralgia, and defines those in which the major surgical operation is to be practised. He confines himself entirely to the consideration of true epileptiform neuralgia, the neuralgia major, or tic douloureux. While examples of it are frequently mistaken in the early stages of the disease, the difficulty can rarely arise when it is well established.

Commenting upon the "chief features" of the disease, Jordan-Lloyd says, with regard to his own cases, that he had twice seen the trouble begin in the first division, and remain most severe there throughout; that spasm of the facial muscles had several times been absent, that there were nearly twice as many females as males, and that he knows of one case of spontaneous disappearance. He points out that epileptiform neuralgia is confined to no class, age, or rank in life. Some of the sufferers are men and women of the highest intellect and the strictest lives. Others have never worked hard, except with their hands; and many have lived freely as regards alcohol and the like. He states that the second division is most often affected, rarely alone, but usually in association with the third division. In about 25 per cent. all three divisions are more or less involved together. He points out, as has been done by all others who have written on the subject, that the disease is rarely of dental origin, and the extraction of healthy teeth is a useless and barbarous method of treatment.

He has no suggestions to make as to the nature and pathology of the disorder, but thinks that it is not improbable that it depends on a true neuritis, but thinks, too, that there are good reasons for

rejecting the theory that vascular degeneration is the causal factor. He disposes of the medicinal treatment by pointing out that only disappointment results from the prolonged administration of drugs, and regards the right treatment as undoubtedly consisting in operation when once the neuralgia is well established. He seems doubtful as to whether the major operation of ganglion excision should be performed in the first instance, or only after the failure of peripheral operations. In operating, he prefers the patient flat on the back, and obviously in a good light. The head and shoulders may be raised to a half-sitting position, or the head may be turned well to the side on a simple sand pillow, without the shoulders being raised. He has tried both positions, and prefers the latter, when a good overhead light is available. He very wisely remarks that a light which enables you to see precisely what you are doing is absolutely necessary. There must be no "plunging in the dark in the neighbourhood of the internal carotid artery and the cavernous sinus." Jordan-Lloyd prefers an osteoplastic flap. He opens the skull by means of a gouge, thinking that it has many advantages over every other instrument. In stripping off the dura from the base of the skull he uses the finger, with its pulp towards the membrane, and takes as his guide the meningeal artery leading to the foramen spinosum. He states that it is necessary to tie and divide the middle meningeal artery close to its exit from this foramen, but many surgeons who have had experience of this operation will not agree with this statement. He says that the ligature of this vessel is "the most tricky part of the operation, and oftentimes taxes the skill and ingenuity of the operator to the utmost." He thinks that removal of the anterior half or more of the ganglion is followed by complete disappearance of the symptoms for which the operation has been performed. He drains the wound with a small rubber tube, laid in its deepest part, and brought to the surface through its hinder angle. He stitches the flap with fine wire or silver wire sutures. He has had no trouble from necrosis of the bone flap.

In his experience, shock after the operation has never been serious, and has usually been absent altogether, and the relief of the painful symptoms has always been immediate and complete. He removes the drainage tube in forty-eight hours, and has been struck by the easy recoveries which follow what appears to be so severe and dangerous an operation. He then passes on to consider the after consequences of the operation, but his observations differ in no detail from those of other observers.

He has operated upon eleven cases without a death. All have been cured of the neuralgic symptoms, and up to the present time there have been no relapses. The length of time since the operation has been as follows:—

Six years and eight months ;  
Four years and ten months ;  
Two years and eleven months ;  
One year and eleven months ;  
One year and nine months ;  
One year and eight months ;  
One year and eight months ;  
Six months ;  
Four months ;

And two others, quite recently.

The average stay in hospital has been a fraction less than twenty-one days. Four of the cases had been submitted to previous operations, one to three, one to two, and two to one operation. The duration of the attacks ranged from two to twenty years, giving an average of nearly nine years. Of the eleven cases, seven were females and four males, and their ages were as follows :—

Three in the fourth decade ;  
Three in the fifth decade ;  
Four in the sixth decade ;  
One in the seventh decade.

The youngest female was forty-six years, and the eldest sixty-seven ; the youngest male fifty-two, and the eldest seventy-three. The right side was affected seven times and the left four. All the males had right-sided lesions, whilst in the females three were on the right and four on the left side.

DONALD ARMOUR.

**EXTIRPATION OF THE GASSERIAN GANGLION.** (*Extirpation (451) des Ganglion Gasseri.*) KRAUSE, *Deut. Gesell. f. Chir.*, April 10, 1901, 33.

IN the writer's experience recurrence of symptoms of neuralgia has frequently occurred after intracranial resection of the two branches of the fifth cranial nerve, and he considers that by the radical operation of extirpation of the root of the fifth nerve and the Gasserian ganglion, in which the risk is scarcely at all increased, a much more certain result is obtained. He had employed this procedure in a series of twenty-seven cases. In one case there was severe collapse, which interrupted the operation, which was completed four days later, but with this exception the operation was completed at one sitting. The patients were usually able to get up on the tenth to the twelfth day, and treatment was discontinued on the eighteenth to twentieth day. In two cases the difficulty of the operation was considerably increased by thinning and perforation of the dura mater, which made it necessary to remove the ganglion in two pieces, but in spite of this he succeeded

in removing the whole of the ganglion, with, however, only the adjacent portion of the fifth nerve. The patient died nineteen days later from influenzal pneumonia, and on section the periosteum of the base of the skull was found to be abnormally thin. The second patient in whom this accident occurred, died of collapse a few hours after the operation (resection of two branches of the fifth nerve). He performed removal of the Gasserian ganglion in a series of twenty-five cases, eighteen of women from 30 to 71, and of seven men from 30 to 72. The right ganglion was removed in twelve women and four men, the left in six women and three men. In connection with the immediate results of the operation he mentions that a delicate woman, who had suffered for years from chronic nephritis, was collapsed for some hours afterwards. A man of 72 died six days after in consequence of heart failure. In this case he had refused to perform the operation on account of the presence of arterial sclerosis and very irregular action of the heart. Post-mortem the wound was found to be closed, and there was no pathological change in its neighbourhood, but there was extensive degeneration of the heart muscle and severe sclerosis. In four patients, who died within a short time of the operation from other complications, the wound was found to be healed on post-mortem examination.

In one case the patient left her bed in eight days with the wound healed, but death ensued four weeks later from cholesteatoma of the brain and its membranes. In no case was a septic process found post-mortem, and the eye, ear and mouth, in spite of their proximity to the wound, remained aseptic throughout. Cerebral abscess has sometimes occurred after several months, but no brain abscess developed in any of the writer's cases, though in two of them the bony plate became necrotic. In one of these intracranial resection of the two branches of the fifth nerve was performed in two sittings, on account of the weakness of the patient; and in the other there was a considerable amount of hæmorrhage, which was accounted for by the fact that the patient had been the subject of hæmophilia. The necrosis manifested itself by œdematous swelling of the cutaneous flaps and their vicinity, especially of the lower eyelid. The wound was enlarged on the tenth day, and further enlarged on the eleventh, and the bony plate, which showed on its inner side a covering of fibropus, was removed. The condition of the eye after operation is important. In some cases, on account of previous peripheral operations, the closing of the lids was rendered impossible by facial paralysis. In one case there was complete lagophthalmus. The Gasserian ganglion was extirpated on August 23, 1895, and up to the present time, though protective measures only were employed, the patient has never suffered from inflammation of

the eye. In another case keratitis appeared three days after operation, soon leading to hypopyon, with purulent affection of the lachrymal sac. He was treated by atropin, lukewarm chloroform water, and a protection bandage, and in spite of the loss of the influence of the fifth nerve this hypopyo-keratitis subsided. His investigations throughout proved that the eyes, in spite of being deprived of the influence of the fifth nerve, recover from hypopyo-keratitis. Trophic disturbance sometimes occurs. In one case ulcers were formed twelve days after operation, with swelling of the adjacent upper lip, which the patient had bitten with her artificial teeth. The bitten portions were removed, and in a few days the swelling disappeared. In another case there was a loss of epithelium on the upper surface of the right side of the tip of the tongue, with a somewhat more extensive one on the inner side of the right lower lip. Boracic lotion was employed, and the tongue healed in five days and the lower lip in nine. Paralysis of the optic muscles were observed in five cases. In one the abducens alone was paralysed, the paralysis disappearing entirely in a few days. Total ophthalmoplegia occurred in a case in which the operation occupied two and a half hours, owing to severe hæmorrhage. The pupil was only moderately enlarged, but reacted neither to light nor accommodation. In ten days a slight degree of functional power was restored in the abducens, and the pupil had become rather smaller. The paralysis gradually improved, but severe inflammation of the cornea occurred three months later. Aphasic disturbances were observed in two cases, and in one case there were severe brain symptoms, and in this case there was an extravasation of blood in the right hemisphere. As regards the results of extirpation, the writer states that none of the patients whom he has operated upon by this method for trigeminal neuralgia, however severe, and who are still living, have up to the present time had a recurrence of symptoms.

DONALD ARMOUR.

---

**REPORT OF THE CONGRESS OF ALIENISTS AND NEUROLOGISTS OF FRENCH-SPEAKING COUNTRIES**

**Held at Lille, August 1906.**

*(Continued.)*

IN his remarkable presidential address, Grasset attributed the great success of this Congress to the fact that it was a collaboration of alienists and neurologists who as a rule tended to be



ignorant of one another's studies, these two branches in general having been entirely separated, not only by the public, but also in the eyes of the medical profession. Both study diseases of the body, and the object of both is to thoroughly understand the normal and morbid functioning of the nervous system, to safeguard society against a progressive invasion by people of abnormal nervous system, and to cure, or at least to solace, those whose nervous system is diseased.

This union is typified in the statues at the gate of the Salpêtrière of Pinel, who reformed the treatment of the insane, and of Charcot, who roused enthusiasm for the study of nervous diseases. Grasset accordingly devoted his remarks to developing the reasons for which he believed in the unity of the science he called human neuro-biology. They are three in number: (1) The same object is studied; (2) the same methods are applied; and (3) the same end is pursued.

1. Although from the definition of the word only psychic diseases should be comprised in the study of psychiatry, yet the psychic functions pertain just as much to the nervous system as to those of motility and sensibility. Moreover, neurologists encounter many psychic phenomena among their patients who have not become insane. Of course Grasset made it clear that by psychic he meant neither occult nor metaphysical phenomena, but those merely into which enter thought and intelligence. These, however, need not be conscious, as say Toulouse, Vaschide, and Piéron. He cited Janet's work on automatism as proving this.

He believes that, although not yet proved, the neurones which preside over the very highest faculties, where one finds sensations, memory, association of ideas, reasoning, and even decision, are not the same as those ruling the phenomena of the inferior psychism, such as are shown in dreams, distraction, hypnotism, somnambule crises, or in mediumistic trances; that with the latter the actor plays his rôle or the reader reads his book, while at the same time with the former he thinks of his fiancée or his financial affairs, just as with these former neurones Archimedes solved his problem without interfering with the automatic reaction of the neurones by which he climbed out of his bath and maintained his equilibrium.

There is no need to enlarge upon "language" in this connection; and although Grasset does not go the length of Renauds, who believes in the psychic individuality of each cellule, yet it is evident that psychic functions are too intimately mingled with other nervous functions to permit of their legitimate separation. Hence as regards their object, psychiatry and neurology are indistinguishable.

2. As to their methods, there is no ground for the suspicion expressed at the Congress of Paris in 1900 that neurologists would

be prevented from thinking anatomically at these reunions; for, as Gilbert Ballet well expressed it, "to think anatomically is no less familiar to psychiatrists." No one who is abreast of the anatomo-pathological work being done in the asylums could make such a statement. That they think physiologically upon occasion is due less to themselves than to the occasion; and Grasset insists upon the necessity of this manner of thought. As says Lepine, "to think anatomically in clinical neurology is superannuated"; with Huchard, "one must for the future think and act physiologically"; and as Albert Robin puts it, "study the diseases of function." Of course he does not deny that an anatomical era was a necessary stage in order to lay the foundation, but he shows that, as regards the determination of the symptoms of a nervous disease, the nature of the lesion is very insignificant as compared with its localisation. No one now says that aphasia is a sign of cerebral softening, amnesia or embarrassed speech a sign of diffuse meningo-encephalitis, paraplegia significative of Pott's disease, or even that dissociation of sensibility is pathognomonic of syringomyelia.

Nor should even these anatomical localities be regarded as the clinical units of which we speak. We shall only escape erroneous descriptions, or at least connotations, by describing the different physiological apparatus which are the real organs, and by refraining from referring the symptoms we find to the organs as described by the anatomist. For instance, the tic of the "colporteur" is meaningless when described in terms of the sterno-mastoid of one side and the trapezius of the other, that is to say of different parts of each spinal accessory nerve whose bond of union in the particular case is the common head-turning centre which controls them both. One occipital lobe presides, not over the other eye, but over the function of seeing objects on the opposite side with both eyes at the same time. The movements of lateral rotation of the eyes are interpreted in the same way; for although anatomists describe the two abducens nerves, each with its centre, yet it is impossible for either of these to act without bringing into action the opposite abducens. That is to say, the true oculo-motor apparatus is not either the third or the sixth pair, but one right-turning nerve and one left-turning nerve for the two eyes. The physiologist and the clinician pre-suppose their existence; it is for the anatomist to reveal them. Thus the physiological right eye is formed by the right half of each eye, and vice versa. Even the nerves of the anatomist are not physiological units. One cannot voluntarily innervate the radial or sciatic nerve to the exclusion of others; the true units are the articulo-motor nerves and the segmento-sensitive nerve. Even the muscles are not clinical unities. For instance, the scapula is raised by one part of the trapezius, while lowered by the other; and the movements

produced by the glutei do not correspond to their anatomical names. But even these do not exist apart; they are merely the terminal portions of the neuro-motor apparatus of motility. The units are not in the muscle or the peripheral topography of the nerves, nor even in the neurones of relays called by the anatomist the real origin; but they are seated in the functional centre, that is the cortex cerebri.

Brissaud has well said of the genio-glossus, "a muscle does not exist: it is an assemblage of fibrils; it is the nerves which determine its function."

Thus, the spinal cord, medulla oblongata, cerebellum, pons Varolii, and brain all disappear as practical entities. There is far more correspondence between the pyramidal tract and the perirolandic zone than between the anterior and posterior columns of the spinal marrow. The real complement of the posterior columns is in the cranium; the real lobes of the brain are not those separated by the fissures, which are in reality bridges rather than ditches.

Following Claude Bernard, for whom the only science in medicine was physiology, we should find in neuro-biology a science of the living being, rather than of the cadaver; and we must cease to study separately the diseases of the brain, those of the pons and the cerebellum, those of the spinal cord, and study instead those of the real organs, *i.e.* those of the apparatus sensitivo-motor, of the apparatus of the psychism, that of orientation and equilibrium, of vision, of hearing, of speech, of circulation, of trophicity, etc.

In each case one must analyse its function in the healthy body, comprehend the abnormalities it shows, observe after death the position of the lesion, and thus deduce the rôle of each part of the nervous system. This method, the only truly fruitful one, is that of both the psychiatrist and the neurologist, no matter upon which part of the nervous system effort is concentrated.

3. The third correspondence of these two branches is their end: (1) to cure, or at least to relieve, those with nervous diseases; (2) to protect society against such patients; and (3) to advance science. Thus, both use the same natural agents—water, mineralised or not; application of heat, etc.; electricity, mechano-therapy—the same drugs, stimulants, tonics, anæsthetics, hypnotics, etc.; their antidotes are directed against the same effects or causes; even psycho-therapy is applied as much by neurologists as by alienists.

Again, the prophylaxis of nervous disease does not differ from that of insanity. For example, bad heredity must be combated, family and social surroundings must be rectified, the personal factor producing neuroses must be regulated—*e.g.* education, overwork, the genital life, the moral habits, professional exigencies, toxic infections; in this it is impossible to separate the alienist and the neurologist.

The practical need of their association is felt even by the magistrates, who, in determination of responsibility, frequently summon both to confer upon the case of a criminal.

Finally, above all, for the better understanding of normal man, each of these workers requires the other. This, after all, is the chief end of medical science, and each practitioner should consider himself primarily a student of human biology, for the sick often teach us to understand the healthy. Philosophers who have studied psychology in asylums for the insane have understood this, and latterly have not neglected the precious data furnished by the non-confined "demi-fous," where they find, ready analysed, the mechanism of emotion, memory, and association.

But there is another reason which alone suffices to bind firmly together these now separated parts of the science. This is the unity of each element which builds up the nervous system. Although the doctrine of the neurone is no longer a purely anatomical one, it yet remains as the physiological unit which alone permits us to explain the phenomena which constitute nervous activity. Does not the section of a nerve influence not only its periphery, but also the proximal portion, and even the cell from which it has grown? Is not a "simple" reflex act almost unheard of in practice? For does not the whole neuronal chain discharge as soon as any one of its constituents is stimulated? Centripetal action involves centrifugal action, and the latter is impossible without the former. The so-called psychic functions are no exception to this rule, and are impossible without movements or their active inhibition, while these in turn influence the psyche; so that it is well said, "sometimes one cries because one is sad, and sometimes one is sad because one cries."

Nor are psychic influences confined to the moving of the so-called "voluntary" muscular system. They influence the secretions, the circulation, and even the trophicity, none of which can be called voluntary.

Thus again is shown the impracticability of arbitrarily dividing into neurology and psychiatry what are indissoluble complements of the physio-pathology of the nervous system of man, or human neuro-biology.

TOM A. WILLIAMS.

*(To be continued.)*

# Bibliography

## ANATOMY

- KRONTHAL. Konstruktionsprinzipien des Nervensystems. *Neurol. Centralbl.*, Okt. 16, 1906, S. 929.
- PETERS. Anatomie und Physiologie des Nervensystems. *Volkskraft-Verlag*, Köln, 1906, M. —40.
- R. B. BEAN. Some Racial Peculiarities of the Negro Brain. *Am. Journ. of Anat.*, Vol. v., No. 4, p. 353.
- JOHN CAMERON. The Histogenesis of Nerve Fibres: a Cytological Study of the Embryonic Cell-Nucleus. *Journ. of Anat. and Physiol.*, Vol. xii., Part 1, p. 8.
- QUENSEL. Beiträge zur Kenntnis der Grosshirnfaserung. *Monatsschr. f. Psychiat. u. Neurol.*, Okt., p. 353.
- K. KROEMER. Die Vereinfachung der Gehirnfaserungsmethode und ihre Verwandbarkeit für den Unterricht. *Anat. Heft*, H. 95, p. 589.
- CARMELO CIACCIO. Sur la reproduction des cellules nerveuses. *Rev. Neurol.*, oct. 15, 1906, p. 876.
- DURANTE. Les transformations morphologiques du tube nerveux (Neuroblaste segmentaire). *Rev. Neurol.*, sept. 30, 1906, p. 836.
- RENATO REBIZZI. Su alcune variazioni delle neurofibrille nella "hirudo medicinalis." *Riv. di Patol. nerv. e ment.*, Vol. xi., f. 8, 1906, p. 355.
- LOOTEN. Recherches anatomiques sur la circulation artérielle du cerveau. *Thèse*. Le Bigot frères, Lille, 1906.
- HALLER. Beiträge zur Phylognese des Grosshirns der Säugetiere. *Arch. f. mikroskop. Anat.*, Bd. 98, H. 1, p. 117.
- BORCHERT. Zur Kenntnis des Centralnervensystems von Torpedo. *Gegenbauers morph. Jahrb.*, Bd. 36, H. 1, p. 52.

## PHYSIOLOGY

- BECKER. Zur Physiologie der Nervenzelle. *Neurol. Centralbl.*, Okt. 1, 1906, S. 382.
- IOTEYKO. Sur l'excitabilité des différents muscles. *Ann. d'Electrobiol. et de Radiol.*, sept. 1906, p. 577.

## PSYCHOLOGY

- W. W. IRELAND. The Psychology of the Crusades. *Journ. of Ment. Sci.*, Oct. 1906, p. 745.
- F. H. BRADLEY. On Floating Ideas and the Imaginary. *Mind*, Oct., p. 445.
- H. FOSTON. The Constitution of Thought. *Mind*, Oct., p. 486.
- HUGH MACCOLL. Symbolic Reasoning. *Mind*, Oct., p. 504.
- ERNST V. ASTER. Beiträge zur Psychologie der Raumwahrnehmung. *Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane*, Bd. 43, H. 3, p. 161.
- AUGUST MESSER. Experimentell-psychologische Untersuchungen über das Denken. *Arch. f. d. gesam. Psychol.*, Bd. 8, H. 1 u. 2, p. 1.
- LOUISE G. ROBINOVITCH. The Genesis of Genius. *Journ. of ment. Pathol.*, No. 5, 1906, p. 209.
- FOREL. L'âme et le système nerveux. Steinheil, Paris, 1906, 5 fr.
- VAILATI. A Study of Platonic Terminology. *Mind*, Oct., p. 473.
- IVAN P. PAWLOW. The Huxley Lecture on the Scientific Investigation of the Psychical Faculties or Processes in the Higher Animals. *Lancet*, Oct. 6, 1906, p. 911.
- CHARLES SCOTT BERRY. The Imitative Tendency of White Rats. *Journ. Comp. Neurol. and Psychol.*, Sept. 1906, p. 333.

- HERRICK. Applications of Dynamic Theory to Physiological Problems. *Journ. Comp. Neurol. and Psychol.*, Sept. 1906, p. 362.  
 HERRICK. Imitation and Volition. *Journ. Comp. Neurol. and Psychol.*, Sept. 1906, p. 376.  
 ALLENGRY. Psychologie et Education. Picard et Kaan, Paris, 1906, 3 fr. 50.  
 KOSTYLEFF. Les Substituts de l'Âme dans la Psychologie moderne. F. Alcan, Paris, 1906, 12 fr.

## PATHOLOGY

- BEVAN LEWIS. The Neuron Theory—Fatigue, Rest, and Sleep. *Journ. of Ment. Sci.*, Oct. 1906, p. 661.  
 MOTT, HALLIBURTON, and EDMUNDS. Regeneration of Nerves. *Proc. Roy. Soc.*, Series B, Vol. lxxviii., No. B 525, p. 259.  
 EGMONT MÜNZER. Das Waller'sche Gesetz, die Neuronlehre und die autogene Regeneration der Nervenfasern. Bd. 7, H. 8, p. 297.  
 LUGARO. Sulla presunta rigenerazione autogena delle radici posteriori. *Riv. di Patol. nerv. e ment.*, Vol. xi., f. 8, 1906, p. 337.  
 JAEGER. Zur Agglutinationsprüfung der Meningokokken. *Wien. med. Woch.*, Oct. 27, p. 2146.  
 POYNTON. A Contribution to the Pathology of Chorea. *Lancet*, Oct. 13, 1906, p. 982.  
 BOLOGNESI. Gliosi cerebrale tuberosa subependimale in soggetto melanconico. *Riv. di Patol. nerv. e ment.*, Vol. xi., f. 8, 1906, p. 348.  
 M. R. PIRONE. L'hypophyse dans la rage. *Arch. de Méd. expér. et d'Anat. path.*, sept., p. 688.  
 G. GUILLAIN et L. ALQUIER. Étude anatomo-pathologique d'un cas de maladie de Dercum. *Arch. de Méd. expér. et d'Anat. path.*, sept., p. 680.  
 HEYDE und CURSCHMANN. Zur Kenntnis der generalisierten metastatischen Carcinose des Zentralnervensystems. *Arb. aus d. Geb. der path. Anat. und Bakt.*, Bd. 5, H. 3, p. 392.  
 DREYFUS. Welche Rolle spielt die Endogenese in der Aetiologie der progressiven Paralyse. *Allg. Ztschr. f. Psychiat.*, Bd. 63, H. 5.

## CLINICAL NEUROLOGY AND PSYCHIATRY

## GENERAL—

- BAILEY. Diseases of the Nervous System resulting from Accident and Injury. Sidney Appleton, London.  
 PURVES STEWART. The Diagnosis of Nervous Diseases. Edward Arnold, London, 1906, 15s.  
 POSEY and SPILLER. The Eye and Nervous System. J. B. Lippincott Co. 25s.  
 WILBRAND und SAENGER. Die Neurologie des Auges. Dritter Band. Zweite Hälfte. Bergmann, Wiesbaden, 1906, 22s. 6d.  
 ASCHAFFENBURG. Die Beziehungen der sexuellen Lebens zur Entstehung von Nerven und Geisteskrankheiten. *Münch. med. Woch.*, Sept. 11, p. 1793.  
 OPPENHEIM. Nervenleiden und Erziehung. Die ersten Zeichen der Nervosität des Kindesalters. Karger, Berlin, 1906, M. 2.  
 FREUD. Sammlung kleiner Schriften zur Neurosenlehre aus den Jahre 1893-1906. Deuticke, Wien, 1906, M. 5.  
 HERMANN EICHHORST. Pathologie und Therapie der Nervenkrankheiten. Urban & Schwarzenberg, Berlin, 1906, M. 9.  
 FÜRNROHR. Die Röntgenstrahlen im Dienste der Neurologie. Karger, Berlin, 1906, M. 10.  
 II<sup>e</sup> Congrès belge de Neurologie et de Psychiatrie, Bruxelles, août 1906. *Journ. de Neurol.*, Nos. 16, 17, 18, 19, 1906.

## MUSCLES—

- ETTORE LEVI. Contributo clinico alla conoscenza del Morbo di Erb-Goldflam. *Riv. di Patol. nerv. e ment.*, Vol. xi., f. 9, 1906, p. 404.  
 MAX KAUFFMANN. Stoffwechseluntersuchungen bei einem Fall von myasthenischer Paralyse. *Monatsschr. f. Psychiat. u. Neurol.*, Okt., p. 299.

GUERMONPREZ. Myopathie par sursauts deltoïdiens pendant les mouvements de l'épaule. *Gaz. des Hôp.*, oct. 11, p. 1383.

GRECO. Supra un caso di assenza congenita del Perone. *La Clinica Moderna*, Sett. 5, p. 424.

#### PERIPHERAL NERVES—

STIEFLER. Zur Klinik der neuralen Form der progressiven Muskelatrophie. *Zachr. f. Heilk.*, Bd. 7, H. 8, p. 219.

LEJONNE et CHARTIER. Névrite ascendante et rhumatisme chronique. *Rev. Neurol.*, oct. 15, 1906, p. 873.

#### SPINAL CORD—

**Tabes.**—RAYMOND. Formes frustes du Tabes. *Bull. méd.*, Vol. xx., No. 64. 1906, p. 625.

GILBERT BALLET. Le Tabes envisagé sous le rapport des Troubles de l'Équilibration. *Journ. de Méd. interne*, mai 1906, p. 121.

MÖRCHEN. Ein kasuistischer Beitrag zu Pals Lehre von den Gefässkrisen der Tabiker. *Neurol. Centralbl.*, Okt. 16, 1906, S. 940.

**Progressive Muscular Atrophy.**—WILLIAM G. SPILLER. Progressive Muscular Atrophy of Cervico-bulbar Type occurring with Cervical Rib. *New York Med. Journ.*, Oct. 6, p. 683.

**Poliomyelitis Anterior Acuta.**—TIEDEMANN. Poliomyelitis acuta und Meningitis cerebrospinalis. *Münch. med. Woch.*, Okt. 23, p. 2095.

**Disseminated Sclerosis.**—MARBURG. Die sogenannte akute multiple Sklerose. Deuticke, Wien, 1906, M. 3.

PETIT et VEILLARD. Paraplégie spasmodique. Troubles cérébraux, sclérose en plaques probable. *Arch. gén. de Méd.*, sept. 25, 1906, p. 2469.

F. T. SIMPSON. A Case of Westphal's Pseudosclerosis. *New York Med. Journ.*, Sept. 29, p. 645.

**Syringomyelia.**—T. K. MONRO. Two Cases of Syringomyelia. *Glasgow Med. Journ.*, Oct., p. 241.

F. CURTIS et L. INGELRANS. Étude sur un cas d'hydro-hématomyelie cervicale traumatique. *Arch. de Méd. exper. et d'Anat. path.*, sept., p. 628.

WILLIAM G. SPILLER. Syringomyelia, extending from the Sacral Region of the Spinal Cord through the Medulla Oblongata, Right Side of the Pons and Right Cerebral Peduncle to the Upper Part of the Right Internal Capsule. *Brit. Med. Journ.*, Oct. 20, 1906, p. 1017.

**Caisson Disease.**—BOINET. La maladie des scaphandriers. *Arch. gén. de Méd.*, sept. 11, 1906, p. 2305.

**Lumbar Puncture.**—MORELLI. Esame del liquido cefalo-rachidiano. *La Clinica Moderna*, Sett. 28, p. 457.

KOPLIK. Percussion of the Skull as a means of placing the Indication for the Performance of Lumbar Puncture, with Special Reference to its Application in Cerebro-spinal Meningitis of the Epidemic Type. *Med. Rec.*, Sept. 29, p. 43.

#### BRAIN—

ANDRÉ LÉRI. Le Cerveau sénile. Le Bigot frères, Lille, 1906.

**Meningitis.**—LINDEMANN. Sind die Steinkohlengruben die Verbreiter der Genickstarre. *Münch. med. Woch.*, Okt. 30, p. 2160.

SIMON FLEXNER. Experimental Cerebro-spinal Meningitis and its Serum Treatment. *Brit. Med. Journ.*, Oct. 20, 1906, p. 1023.

AMBLARD. Méningite cérébro-spinale à pneumocoques et diabète. *Arch. gén. de Méd.*, sept. 11, 1906, p. 2319.

**Encephalitis.**—KUTSCHER. Aetiologie und Epidemiologie der übertragbaren Gehirnhautentzündung. *Berlin. klin. Woch.*, Okt. 8, p. 1344.

**Tumour.**—RAYMOND. I. Un cas simple de tumeur de l'encéphale (Localisation cérébelleuse). II. Un cas complexe avec troubles mentaux accentués et primitifs (Localisation sur le corps calleux). *Arch. gén. de Méd.*, oct. 2, 1906, p. 2527.

MACEWEN. A Case of Cerebral Tumour, giving rise to Jacksonian Epilepsy, and at a later stage, Coma; Operation; Removal of Tumour; Recovery. *Lancet*, Oct. 13, 1906, p. 992.

MAILLARD et MILHIT. Un cas de tumeur cérébrale avec sommeil. *L'Encéphale*, Vol. i., No. 3, 1906, p. 230.

- BABONNEIX. Les kystes hydatiques du cerveau chez l'enfant. *Rev. mens. des Maladies de l'Enfance*, sept., p. 385.
- Abcess.**—OBERNDÖRFFER. Zur Differential-diagnose otitischer und metastatischer Hirnabscesse. *Deutsch. med. Woch.*, Okt. 4, p. 1617.
- Sinus Thrombosis.**—ADOLPH BRONNER. Notes on an Unusual Case of Thrombosis of the Lower Part of the Lateral Sinus, of Aural Origin. *Lancet*, Oct. 27, 1906, p. 1142.
- Amaurotic Family Idiocy.**—HUISMANS. Ein Fall von Tay-Sach'scher familiärer amaurotischer Idiotie. *Deutsch. med. Woch.*, Okt. 25, p. 1737.
- General Paralysis.**—HEILBRONNER. Frühdiagnose und Behandlung der progressiven Paralyse. *Deutsch. med. Woch.*, Okt. 4, p. 1609.
- Cerebellum.**—DANA. Functions of the Cerebellum and the Symptoms of its Disease. *New York Med. Journ.*, Oct. 6, p. 677.
- BERNHEIM. Pathogénie d'un cas d'hystérie liée à une tumeur cérébelleuse. *Bull. méd.*, Vol. xx., No. 52, 1906, p. 603.
- GARBINI. Tubercoli multipli del cervello interessanti il nucleus caudatus, il præcuneus ed il lobulus quadrangularis. *Manicomio*, Anno xxii., N. 2, 1906, p. 187.
- A. V. M. ANDERSON. Case of Cerebellar Hæmorrhage. *Inter-Colonial Med. Journ. of Australia*, Aug. 20, p. 439.

#### MENTAL DISEASES—

- ROBERT JONES. The Evolution of Insanity. *Journ. of Ment. Sci.*, Oct. 1906, p. 629.
- NOLAN. The Possibility of the Limitation of Lunacy by Legislation. *Journ. of Ment. Sci.*, Oct. 1906, p. 756.
- OLIVA. Nota e critica di tecnica manicomiale. *Manicomio*, Anno xxii., N. 2, 1906, p. 276.
- JAMES J. PUTNAM. The Bearing of Philosophy on Psychiatry, with Special Reference to the Treatment of Psychasthenia. *Brit. Med. Journ.*, Oct. 20, 1906, p. 1021.
- MÖNKEMÖLLER. Geisteskrankheit und Geistesschwäche in Satire, Sprichwort und Humor. Marhold, Halle, 1906, M. 6.
- CAMPBELL MEYERS. The Pre-Insane Stage of Acute Mental Disease. *Brit. Med. Journ.*, Oct. 20, 1906, p. 1032.
- GEORGES DREYFUS. Ein Beitrag zur Kenntnis des hysterischen Irreseins. *Centralbl. f. Nervenheilk. u. Psychiat.*, Okt. 1906, S. 785.
- J. S. BOLTON. Amentia and Dementia. *Journ. of Ment. Sci.*, Oct. 1906, p. 711.
- FRANCESCO BURZIO. Idiozia ed atetosi doppia. *Ann. di Freniatria*, Vol. xvi., f. 3, 1906, p. 217.
- FRANCESCO BURZIO. Ricerche antropologiche sul cretinismo. *Ann. di Freniatria*, Vol. xvi., f. 3, 1906, p. 238.
- WILMANNS. Zur Psychopathologie des Landstreichers. Barth, Leipzig, 1906, M.—50.
- A. MARIE. Mysticisme et Folie. Giard et Brière, Paris, 1906, 6 fr.
- CLARENCE B. FARRER. Types of the Devolutional Psychoses. *Rev. Neurol. and Psychiat.*, Oct. 1906, p. 665.
- WILMANNS. Die leichten Fälle des manisch-depressiven Irreseins (Zyklothymie) und ihre Beziehungen zu Störungen der Verdauungsorgane. Breitkopf & Härtel, Leipzig, 1906, M.—75.
- PETERS. Die Kulturkrankheit. Wirkungen und Folgen der sinnl. Fehler. Chronische Krankheiten, Nervosität, etc. *Volkskraft-Verlag*, Köln, 1906, M.—60.
- BARUK. Les Troubles mentaux de la grossesse et de l'état puerpéral. Germain et Grassin, Angers, 1906.
- LENER. Sulla origine emotiva delle malattie della volontà. *Manicomio*, Anno xxii., N. 2, 1906, p. 163.
- EMIL AMBERG. Ear Affections and Mental Disturbances. *Journ. Nerv. and Ment. Dis.*, Oct. 1906, p. 651.
- CHARLES MERCIER. Agoraphobia—a Remedy. *Lancet*, Oct. 13, 1906, p. 990.
- PFFERSDORFF. Ueber Stereotypieen im manisch-depressiven Irresein. *Centralbl. f. Nervenheilk. u. Psychiat.*, Okt. 1, 1906, S. 745.
- RÉGIS. La Poésie dans les Maladies Mentales. *L'Encéphale*, Vol. i., No. 3, 1906, p. 262.
- MARCHAND. Stéréotopie graphique chez un dément précoce. *Journ. de Neurol.*, oct. 20, 1906, p. 529.



- SCHRÖDER. Beitrag zur Lehre von den Intoxikationspsychosen. *Allg. Zeit. f. Psychiat.*, Bd. 63, H. 5.
- LAPINSKY. Über Psychosen nach Augenoperationen. *Allg. Zeit. f. Psychiat.*, Bd. 63, H. 51.
- C. C. EASTERBROOK. Insanity and Indicanuria. A Note of Criticism. *Journ. of Ment. Sci.*, Oct. 1906, p. 766.
- STELZNER. Analyse von 200 Selbstmordfällen nebst Beitrag zur Prognostik der mit Selbstmordgedanken verknüpften Psychosen. Karger, Berlin, 1906, M. 4.
- ROSSI. Sullo stato mentale di Ottavia Silva. Parere freniatrico in causa d'interdizione. *Manicomio*, Anno xxii., N. 2, 1906, p. 178.
- TIRELLI. Perizie medico-legale sulle condizioni mentali di Rosa Bonetti (cont.). *Ann. di Freniatria*, Vol. xvi., f. 3, 1906, p. 241.
- DIDE. Étude cytologique, bactériologique et expérimentale du sang chez les aliénés. Le Bigot frères, Lille, 1906.
- LIEFMANN und NETER. Ueber Ruhr bei Irren. *Münch. med. Woch.*, Okt. 23, p. 2097.
- WERNER. Die Versorgung der geisteskranken Verbrecher in Dalldorf. Fischer, Berlin, 1906, M. 4.
- EDWIN GOODALL. An Address on the Hospital Treatment of Curable Cases of Mental Disorder. *Brit. Med. Journ.*, Oct. 27, 1906, p. 1084.

#### ALCOHOLISM—

- DAVID FLECK. Inebriety and Mental Weakness. *Brit. Journ. Inebriety*, Oct. 1906, p. 99.
- WEBER. Alcohol and Old Age. *Brit. Journ. Inebriety*, Oct. 1906, p. 87.
- HORSLEY. The Relations of Inebriety and Crime. *Brit. Journ. Inebriety*, Oct. 1906, p. 66.
- ISADOR H. CORIAT. Mental States in the Subjects of Alcoholic Neuritis. *Brit. Journ. Inebriety*, Oct. 1906, p. 106.
- TOMASCHY. Ueber Alkoholversuche bei Beurteilung zweifelhafter Geisteszustände. *Allg. Zeit. f. Psychiat.*, Bd. 63, H. 5.
- MOTT. Alcohol and Insanity. The Effects of Alcohol on the Body and Mind as shown by Asylum and Hospital Experience in the Wards and Post-mortem Room. *Journ. of Ment. Sci.*, Oct. 1906, p. 678.

#### GENERAL AND FUNCTIONAL DISEASES—

- Epilepsy.**—VOLLAND. Geburtstörungen und Epilepsie. *Allg. Zeit. f. Psychiat.*, Bd. 63, H. 5.
- T. DIVINE. A Study of the Mortality from Convulsions in the "Rickets Group" of Affections in Infancy. *Brit. Journ. of Child. Dis.*, Oct., p. 443.
- URBANTSCHITSCH. Ueber "Reflexepilepsie." *Wien. klin. Woch.*, Sept. 27, p. 1160.
- STOWELL. One hundred and three Cases of Epilepsy. *Med. Rec.*, Sept. 29, p. 490.
- BURR. A Case of Myoclonus Epilepsy with Autopsy. *New York Med. Journ.*, Sept. 29, p. 625.
- N. B. ROSS. A Note on Peculiar Attitudes in Epilepsy during Sleep. *New York Med. Journ.*, Oct. 6, p. 689.
- BARUK. Traitement de l'épilepsie par les bromures et l'hypochloruration. Germain et Grassin, Angers, 1906.
- J. VOISIN, R. VOISIN, et J. RENDU. Nouvelles recherches sur les traitements de l'épilepsie par la bromuration avec ou sans déchloruration. *Arch. de Neurol.*, sept., p. 161.
- Neurasthenia.**—LE CLECH. De la neurasthénie grave d'origine osseuse. Des modes réactionnelles du système nerveux. Les réflexes de défense dans les guérisons naturelles. Impr. nationale, Paris, 1906.
- CESARE BIONDI. Sulla Sintomatologia e sulla Patogenesi delle neurosi traumatiche. *Ann. di Freniatria*, Vol. xvi., f. 3, 1906, p. 193.
- HUGH A. MACCALLUM. Gastric Neurasthenia. *Brit. Med. Journ.*, Oct. 20, 1906, p. 1031.
- BATUAUD. La neurasthénie génitale féminine. Maloine, Paris, 1906, 4 frs.
- MENNELLA. Neurastenia e auto-intossicazioni. *Manicomio*, Anno xxii., N. 2, 1906, p. 209.
- ZORN. Zur Behandlung der Neurasthenie sexualis. Leipzig, 1906, M.—50.

- Hysteria.**—DUXIAN. Sur deux cas de fièvre hystérique. *Rev. méd. de la Suisse Romande*, sept. 20, p. 490.  
 LEROY. La Responsabilité des hystériques. Le Bigot frères, Lille, 1906.  
 MORTON PRINCE. Hysteria from the Point of View of Dissociated Personality. *Boston Med. and Surg. Journ.*, Oct. 4, p. 372, and Oct. 11, p. 407.  
**Tetany.**—VAN PEE et LARUELLE. A propos d'un cas de tétanie. *Rev. d'Hygiène et de Méd. infant.*, T. 5, Nos. 4 and 5, p. 447.  
**Neuralgia.**—LEVY. Essai sur les neuralgies faciales. *Thèse*. Roussel, Paris, 1906.

#### SPECIAL SENSES AND CRANIAL NERVES—

- WEYL. Ueber Nystagmus toxicus. *Berlin. klin. Woch.*, Sept. 17, p. 1245.  
 MAX LÖWY. Ueber die Schmerzreaktion der Pupillen als ein differential-diagnostisches Zeichen zwischen organischer und psychogener Druckschmerzhaftigkeit. *Neurol. Centralbl.*, Okt. 16, 1906, S. 947.

#### MISCELLANEOUS SYMPTOMS—

- R. RICHARD. Überblick über den heutigen Stand der Frage nach der Lokalisation in der Grosshirnrinde und ihre Anwendung in der forensischen Praxis. *Monatsschr. f. Psychiat. u. Neurol.*, Okt., p. 331.  
 PONTHEU. Contribution à l'étude de la pseudo-paralysie rachitique. *Thèse*. Le Bigot frères, Lille, 1906.  
 ESPOSITO. Amiotrofia da trauma nervoso periferico (cont.). *Manicomio*, Anno xxii., N. 2, 1906, p. 233.  
 C. K. MILLS and T. H. WEISENBURG. The Subdivision of the Representation of Cutaneous and Muscular Sensibility and of Stereognosis in the Cerebral Cortex. *Journ. Nerv. and Ment. Dis.*, Oct. 1906, p. 617.  
 DUPRAY. Section incomplète du nerf cubital avec lésions paralytiques et trophiques et persistance de la sensibilité. *Rev. méd. de la Suisse Romande*, Sept. 20, p. 477.  
 JOSEF FEIX. Ueber ein neues Verfahren zur Untersuchung des Patellar- und Achilles-Sehnenreflexes. *Wien. klin. Woch.*, Okt. 11, p. 1223.  
 KRÖNIG. Ein einfacher Kunstgriff zur Erzeugung des Kniephänomens. *Berlin. klin. Woch.*, Okt. 29, p. 1421.  
 BOTTEMER. Le Réflexe crémasterien chez les hernieux et à la suite des cures radicales de hernies du réflexe crémasterien. (*Thèse*.) Impr. réunies, Lyon, 1906.  
 CLAUDE et ROSE. Etude graphique du clonus dans les maladies organiques et fonctionnelles. *Rev. Neurol.*, sept. 30, 1906, p. 829.  
 REISSERT. Beitrag zur Kenntnis der Lidreflexe. *Klin. Mon. f. Augenheilk.*, Okt., p. 378.  
 ANDRÉ SOLIRÈNE. Les complications nerveuses des appendicites. *Thèse*. Bonvalot-Jouve, Paris, 1906.  
 HERMANN SCHLESINGER. Ueber die paroxysmale Tachykardie und ihre Beziehungen zu den Erkrankungen des Nervensystems. *Sammlung klin. Vorträge innere Medizin*, No. 131.  
 SOLLIER. La claudication intermittente de la moelle. *Presse méd.*, oct. 24, p. 677.  
 GUSTAV MUSKAT. Ueber das intermittierende Hinken. *Zeit. f. orthopäed. Chirurg.*, Bd. 16, H. 1 u. 2, p. 184.  
 VOGT. Fälle von familiärer Mikrokephalie. *Allg. Zeit. f. Psychiat.*, Bd. 63, H. 5.  
 HRUSKA. Ein Fall von Typhus abdominalis mit nachfolgender Lähmung und Aphasie. *Prag. méd. Woch.*, Sept. 20, p. 487.  
 PIERRE MARIE. Revision de la question de l'aphasie: que faut-il penser des aphasies sous-corticales (aphasies pures)? *Sem. méd.*, oct. 17, p. 493.  
 VARIOT et LECOMTE. Un cas de typhlolexia congénitale (cécité congénitale verbale). *Gaz. des Hôp.*, oct. 3, p. 1479.  
 GUTZMANN. Untersuchungen über die Grenzen der sprachlichen Perzeptionen. *Zeit. f. klin. Med.*, Bd. 60, H. 3 u. 4, p. 233.

#### TREATMENT\*

- L. F. BARKER. Some Experience with the Simpler Methods of Psychotherapy and Re-education. *Am. Journ. of the Med. Sci.*, Oct., p. 499.  
 HUDOVERNIG. Die Verwendbarkeit des Methylnatrinum bromatum bei Erkrankungen des Nervensystems. *Berlin. klin. Woch.*, Okt. 15, p. 1363.

\* A number of references to papers on Treatment are included in the Bibliography under the individual Diseases.

- E. GROSSMANN. Die Behandlung der Ischias mit perineuraler Kochsalz-infiltration. *Wien. klin. Woch.*, Okt. 18, p. 1254.
- LAPPONI. L'Hypnotisme et le Spiritisme. Étude médico-critique. Perrin et Cie, Paris, 1906, 3 fr. 50.
- ZIMMERN. Éléments d'Électrothérapie clinique. Masson et Cie, Paris, 1906.
- SUDNIK. Étude clinique de l'influence de la durée de l'onde électrique sur les effets moteurs et sensitifs. *Ann. d'Electrobiol. et de Radiol.*, sept. 1906, p. 610.
- W. W. GRAVES. The Problem of Localisation in Relation to Head Injuries. *Med. Rec.*, Sept. 29, p. 483.
- MOSCHCOWITZ. The Surgical Treatment of Trigeminal Neuralgia. *Med. Rec.*, Sept. 29, p. 486.
- HAYNES. Gunshot Wounds of the Spinal Cord. *New York Med. Journ.*, Sept. 29, p. 629.
- SPILLER and FRAZIER. Cerebral Decompression. Palliative Operation in the Treatment of Tumours of the Brain. *Journ. of Am. Med. Assoc.*, Sept. 22, p. 923.
- ALLEN STARR. The Present Status of Brain Surgery. *Journ. of Am. Med. Assoc.*, Sept. 22, p. 926.
- HENKING. Beitrag zur chirurgischen Behandlung schwerer Occipital- und Cervicalneuralgien. *St Petersburg. med. Woch.*, Sept. 7 (20), p. 391.
- VULPIUS. Misserfolge der Sehnenüberpflanzung. *Berlin. klin. Woch.*, Okt. 15, p. 1359.
- ALT. Ein Beitrag zur operativen Behandlung der otogenen Fazialis-lähmung. *Wien. klin. Woch.*, Okt. 25, p. 1285.
- WEISENBURG. Advances in the Surgery of the Nervous System considered from the Standpoint of the Neurologist. *New York Med. Journ.*, Oct. 13, p. 732.
- F. W. MURRAY. Early Operation in Traumatic Intracranial Hæmorrhage. *Annals of Surgery*, Sept. 1906, p. 374.
- SHERREN. Some Points in the Surgery of the Peripheral Nerves. *Edin. Med. Journ.*, Oct., p. 297.
- R. T. WILLIAMSON. Recent Successful Results of the Operative Treatment for Spinal Tumours and Cysts. (Review.) *Med. Chronicle*, Oct., p. 23.

#### BOOKS AND PAMPHLETS RECEIVED.

- Stewart, Purves. "The Diagnosis of Nervous Diseases." Edward Arnold, London, 1906.
- Clouston, T. S. "The Hygiene of Mind." Methuen & Co., London, 1906.
- Zimmern, A. "Éléments d'Électrothérapie Clinique." Masson et Cie, Paris, 1906.
- Fürnrohr, Wilhelm. "Die Röntgenstrahlen im Dienste der Neurologie." Karger, Berlin, 1906.
- Stelzner, Helenefriederike. "Analyse von 200 Selbstmordfällen." Karger, Berlin, 1906.
- Babes, Victor. "Atlas der pathologische Histologie des Nervensystems." August Hirschwald, Berlin, 1906.
- Haskovec. "Revue v. Neurologii, Psychiatrii, fysikalni a diaeteticke Therapii." Prag, 1906.
- Rentoul, Robert Reid. "Race Culture or Race Suicide." Walter Scott Publishing Co., Ltd., London, 1906.

# Review of Neurology and Psychiatry

---

## Original Articles

### **A NOTE UPON TWO IMPORTANT POINTS IN THE LOCALISATION OF TUMOURS OF THE FRONTAL REGION OF THE BRAIN.**

By T. GRAINGER STEWART, M.B. Ed., M.R.C.P. Lond.,  
Assistant Physician to the Metropolitan Hospital, Pathologist to the National  
Hospital for the Paralysed and Epileptic.

AMONGST the various localising signs presented by tumours of the frontal lobes of the brain are two to which I would like to draw special attention. The first is the absence, diminution, or easy exhaustion of the superficial abdominal reflexes on the side opposite to the tumour; the second is the occurrence of a fine, rapid vibratory tremor in the limbs of the same side as the tumour.

Both these signs are often present in cases of frontal tumour, and the diminution of the superficial abdominal reflexes is rarely absent in any case in which the growth has attained such a size as to give rise to the general symptoms of intracranial neoplasm.

Loss or diminution of the superficial abdominal reflexes, local causes being excluded, would appear to depend on some affection of the pyramidal system. Such alteration of these reflexes is seen on the hemiplegic side in cases of hemiplegia, and on both sides in cases of spastic paraplegia, due to lesions above the mid-dorsal region. This loss of the superficial

abdominal reflexes is usually associated with an increase in the corresponding deep reflexes, and the presence of an extensor plantar response. In cases of cerebral tumour the alteration of the abdominal reflexes occurs before any change may be noted in the deep reflexes, or in the plantar response, and has often been observed to precede the other signs, reflex or motor, of hemiplegia. It is therefore of value as a localising sign in all cases of cerebral tumour; but in cases of tumour of the frontal region, where focal symptoms are often late in appearing, it is of special service. I have observed it as the first localising sign in several cases of tumour of the frontal lobes, and I have never known it to prove fallacious. In certain cases where the intracranial tension is much increased, or where there is a growth in both frontal lobes, the superficial abdominal reflexes may be absent on both sides; but as a rule when this occurs an extensor plantar response will be obtained on the side contra-lateral to the tumour, so that any doubt as to the side of the lesion can be dispelled.

The second sign—tremor in the limbs on the same side as the tumour—may be observed in both the upper and lower limbs, but it is more constant and better seen in the arm than in the leg. It is absent during muscular rest, and is best brought out by making the patient extend both arms horizontally in front of him with the palms directed downwards and the fingers extended. It will then be noticed that the homolateral arm and hand are in a state of constant fine vibratory tremor in contrast to the contralateral arm, in which such tremor is absent. The difference between the two hands can be better realised by placing a palm lightly upon the back of each of the patient's hands. This tremor may not be constantly present in any one case, but in the great majority of cases of frontal tumour it will be observed at one time or another. During the past four years I have had the opportunity of studying at the National Hospital for the Paralysed and Epileptic more than twenty cases of tumour of the frontal lobes, and in all of these except two, in which the patients' condition prevented examination for this point, it was present at one time or another. In five cases out of twenty-two such tremor was observed on both sides, but in all it was greater and more constant on the side of the tumour. Of these five cases, in which bilateral tremor was observed, in two

TABLE.

Side of Tumour.	No.	Case.	Sides.	Nature of Growth.	Motor Paresis.		Tremor.	Reflexes.			
					Face alone.	Hemi-paresis.		Deep.	Abdominal.	Plantar.	
Right	1	W. M.	right left	gumma	O O	O O	tremor O	N +	N O	flexor extensor	
Right	2	A. O.	right left	gumma -	O slight	O O	tremor O	N +	N -	flexor extensor	
Right	3	T. J.	right left	glioma -	O slight	O O	tremor O	+	N O	flexor ? flexor	
Right	4	W. M.	right left	blood cyst -	O slight	O O	tremor O	+	N O	flexor flexor	
Right	5	C. T.	right left	glioma -	O slight	O O	tremor O	N N	- O	flexor flexor	
Right	6	J. D.	right left	carcinoma -	O marked	O O	tremor O	N N	N O	flexor flexor	
Right	7	W. M.	right left	glioma -	O marked	O slight	tremor O	N N	- O	flexor flexor	
Right	8	J. C.	right left	glioma -	O marked	O slight	tremor O	N +	N O	flexor flexor	
Left	9	E. B.	right left	- glioma	O O	O O	O tremor	+	O N	flexor ? flexor	
Left	10	H. B.	right left	- endothelioma	slight O	O O	O tremor	N N	N O	flexor flexor	* Had occasional tremor on left as well.
Left	11	E. R.	right left	- endothelioma	slight O	O O	slight tremor	N N	N -	flexor flexor	* Tremor afterwards only seen on left.
Left	12	E. J.	right left	- glioma	O O	O O	O tremor	N N	O N	flexor flexor	
Left	13	E. P.	right left	fibroma	slight O	O O	O tremor	+	O -	extensor flexor	
Left	14	J. M.	right left	- glioma	slight O	O O	O O	N N	O N	extensor flexor	* Tremor not examineded for.
Left	15	W. Y.	right left	- glioma	slight O	slight O	O tremor	+	O N	extensor flexor	
Left	16	A. D.	right left	- glioma	marked O	slight O	O tremor	+	O N	? flexor flexor	
Left	17	S. B.	right left	- sarcoma	marked O	marked O	O O	+	O N	extensor flexor	* Tremor not looked for.
Left	18	S. H.	right left	- glioma	marked O	marked O	O tremor	+	O N	? flexor flexor	
Both	19	J. H.	right left	glioma glioma	O O	O O	tremor tremor	N N	N ? N ?	flexor flexor	* Both lobes involved.
Both	20	F.	right left	endothelioma endothelioma	slight O	slight O	tremor tremor	+	O -	? extensor flexor	* Left lobe more involved than right.

Abbreviations: N=natural; O=absent; - =diminished; + =increased.

autopsy revealed the presence of tumours in both frontal lobes. This tremor occurs quite independently of paresis of the other (the contra-lateral) side, and there does not appear to be any association between them. I have specially looked for similar tremor in cases of tumour situated in other parts of the brain, but have never observed it. Various forms of tremor and unsteadiness are not uncommonly met with in association with cerebral neoplasms ; and although some of them approach the type I have endeavoured to describe, yet as a rule they lack the fine vibratory character.

The table shows the condition of the motor system and reflexes in twenty cases of tumour or gross lesion of the frontal lobes of the brain. In each instance, except in the two cases in which tremor was not observed, the condition reported is that which was found when the tremor was first observed.

These cases were all observed in the National Hospital for the Paralysed and Epileptic, and I am indebted to the kindness of the medical staff of the hospital for permission to make use of them.

It will be seen from the Table that—

- (1) Diminution or loss of the superficial abdominal reflexes was observed on the contra-lateral side in every case except one (19).
  - (2) The corresponding plantar reflexes were extensor in 7, doubtful in 4, and flexor in 9, of the 20 cases.
  - (3) Tremor was noted on the same (homolateral) side as the tumour in 18 out of the 20 cases.
  - (4) That it was present on both sides in the 2 cases (10 and 11) where the growth was limited to one side, and that it was present on both sides in the two cases where the growth was bilateral (19 and 20).
-

**NOTE ON A CASE OF JUVENILE GENERAL PARALYSIS ;  
ABSENCE OF STIGMATA OF CONGENITAL SYPHILIS  
AND OF A FAMILY HISTORY INDICATIVE OF THAT  
DISEASE ; VERY PRONOUNCED CEREBRO-SPINAL  
LYMPHOCYTOSIS.**

By EDWIN BRAMWELL, M.B., F.R.C.P.E., F.R.S.E., M.R.C.P. Lond.,  
Assistant Physician to Leith Hospital.

SINCE 1876, when the first recorded case of Juvenile General Paralysis was described by Clouston,<sup>1</sup> a large number of instances have been reported, while the existence of an important etiological relationship between syphilis and these cases of general paralysis occurring in early life has long been recognised. Thiry, for example, obtained certain or extremely probable evidence of syphilis, either congenital or acquired, in 43 of 67 cases which he collected from the literature, while in only 10 of these cases were there no indications suggestive of that disease.<sup>2</sup> Further, it is to be remembered that syphilis cannot be absolutely excluded in the absence of manifestations which might serve for its detection, hence it is not improbable, as Thiry remarks, that the disease may have been present even in some of those cases in which no evidence of its existence was forthcoming.

The following case, for example, presented none of the stigmata of congenital syphilis, while an inquiry into the family history revealed no facts distinctly indicative of syphilis in the parents ; yet examination of the cerebro-spinal fluid showed a very pronounced lymphocytosis, an observation which must be regarded as pointing strongly to the syphilitic origin of the nervous affection.

**RECORD OF CASE.**

J. K., aged 15½, was seen in the out-patient department of Leith Hospital on June 26th, 1906. His mother, who came with him, stated that her son was very nervous and shaky, that his speech was indistinct and his memory defective.

*Previous Health.*—The following history was obtained from the patient's

<sup>1</sup> *Journ. of Ment. Science*, Oct. 1877, p. 419.

<sup>2</sup> *De la Paralyse générale progressive dans la jeune âge*, p. 87. Paris, 1898.



mother, a particularly intelligent woman. J. K. was her first child, and was born at full time after a comparatively easy labour which did not necessitate the use of instruments. The patient appeared to be a perfectly healthy child at birth, and, so far as can be ascertained, did not subsequently suffer from snuffles, nor was any skin rash noticed. He was breast fed up to the age of six months. As far as his mother remembers, he cut his first teeth at about the usual time, and began to walk when he was 16 months old. Although he was a little backward in beginning to talk, he commenced all of a sudden to speak correctly without having passed through the stage of baby language, so that by the time he was two years old he was talking as well as most children of that age. Measles, followed by bronchitis, when he was between 3 and 4 years old, and scarlet fever seven years later, which appears to have run a normal course, were the only illnesses from which he had suffered. He went to school when 5 years of age and remained there until he was 14. He was always rather backward for his age, and when he left he was only in the fourth standard.

*Present Illness.*—The present illness dated from February 1905 (sixteen months previous to examination), when he was knocked over by a runaway horse. He was said to have injured his right knee, and to have been unable to walk in consequence for five weeks, during most of which time he was confined to bed. There was no evidence to suggest that his head received any direct injury as a result of the accident. The patient, his mother stated, had received a considerable "nervous shock," and soon after the accident he lost his speech for some days. Ever since that time he had spoken indistinctly. A few weeks after the accident he was observed to be shaky, while about the same time his memory was noticed to be impaired. These symptoms seemed to have developed gradually, so that it was impossible to fix any definite date for their onset. His mother stated, however, most emphatically, upon cross-examination, that none of these symptoms were present before the accident, but that they began to develop almost immediately after. On two occasions soon after the accident he is said to have fallen out of bed in what appeared to be a fit. He was unconscious for a few minutes, but does not seem to have been convulsed. In April 1906, he had an attack in which for a few minutes he lost consciousness, and became stiff all over, while his face was very blue. During the two or three months which immediately preceded our examination his condition had been becoming worse. He had been very irritable and easily upset, though easily managed. When sent a message he often forgot what he had been sent for. He was in the habit of wandering about collecting small pieces of wood, nails, and other odds and ends with which he filled his pockets. He was rather particular about his clothes, more so than formerly. He was very careful about his money, putting every penny he received into a money-box which he carried about with him and liked to rattle, and nothing would induce him to spend a penny.

*State upon Examination.*—The patient's somewhat stupid, staring, and at the same time scared expression, at once attracted attention. There was very slight double ptosis and marked wrinkling of the forehead (frontal over-

action). He was somewhat poorly developed for his age, though not ill-nourished. There were no distinct stigmata of degeneracy, nor were there any signs of congenital syphilis. The nose was well formed, the forehead was not unduly prominent, the cornea of both eyes was quite clear; there were no scars about the mouth, and the teeth presented none of the characteristics described by Mr Jonathan Hutchinson. The genitalia were poorly developed, and there was no trace of pubic or axillary hair.

When spoken to his face showed no expression of responsive intelligence, and he turned to his mother as if expecting her to answer for him. His manner was childish, and he was unduly emotional. Although not word-deaf the patient obviously took a long time to grasp the meaning of a question. On being told to open his eyes widely he opened his mouth, and it took a few minutes before he could be made to realise that this was not what was required of him. He remembered the number of the house and the name of the street in which he lived, but when questioned as to the number, names, and ages of the boys who were in his class at school, his feeble memory was at once apparent. His mother stated that he could find his way about the streets quite well.

When he spoke there was a great deal of tremor of the face, both of the lips and eyelids; his voice was rather high-pitched and monotonous, and his articulation indistinct and slurring. For hippopotamus, for instance, he said "hip-popoto-potopos," while West Register Street was pronounced "Wesh Regissherr Shhreeshh." His writing was very tremulous, although when asked to write his name he spelt it correctly.

Vision seemed to be good, for the patient was able to make out small type, though when asked to read a simple sentence he did so in such a way as to leave the impression that it conveyed no meaning to him. The exact acuity of vision was not tested either with Snellen or Jaeger types. The optic discs presented a healthy appearance, an observation which was confirmed by Dr Sinclair, who noted also the absence of any choroiditis.

A watch was heard at several inches from either ear.

As regards the eye muscles, a very slight degree of double ptosis was present, and associated with this was marked frontal overaction. There was no squint. The ocular movements, with the exception of convergence, which was not satisfactory, were unimpaired, and there was no nystagmus.

The pupils were somewhat large and equal (measuring about 6 mm.); they were regular in outline, were quite immobile to light, and only contracted very slightly on accommodation. As already mentioned, convergence was extremely defective.

With the exception of the unsteadiness of the face, which has already been referred to, nothing abnormal was detected in connection with the other cranial nerves, excepting for the fact that the tongue was extremely tremulous.

The grasp was rather poor but equal. Both hands were extremely tremulous, the right rather more so than the left. His mother stated that he was unable to feed himself with the right hand on account of the shakiness. When he closed the eyes he missed the tip of the nose by an inch or two with the right forefinger, whereas with the left he performed this test fairly accurately. No definite weakness of any group of muscles in either lower

limb or trunk was detected. He walked somewhat unsteadily in rather a shuffling manner and with short steps. There was no Rombergism.

The knee- and ankle-jerks were brisk and equal. There was no ankle clonus. The right plantar reflex was distinctly of the flexor type, while that on the left side was noted as "indeterminate." No anæsthesia or analgesia was detected. The calf muscles were not analgetic.

There was no defect of micturition so far as could be ascertained.

The heart was not enlarged, the sounds were closed in all the areas, and the pulse was regular.

The bowels were regular.

The urine contained neither albumin nor sugar.

On lumbar puncture there was no evidence of increased pressure. The fluid obtained was perfectly clear. A cytological examination showed on two separate occasions the presence of a very marked lymphocytosis, 120 to 150 lymphocytes being present in many fields under a magnification of 400 diameters.

*Family History.*—The patient's father has been an alcoholic for many years, and was drinking heavily about the time of patient's conception. No history of syphilis was obtained from the mother. The parents, who were not blood relations, had had five children, of whom two were alive. The patient was the eldest. Then followed two children who both died at three months of diarrhœa and vomiting. The next child, who was aged eleven years, presented on examination no signs of congenital syphilis. The last child died when three weeks old of diarrhœa and vomiting. There had been no miscarriages.

It is unnecessary to comment further on the case. The diagnosis, as will be seen from the above record, was obvious. Again there was no evidence of syphilis either congenital or acquired, while lastly the cerebro-spinal fluid obtained by lumbar puncture showed an excessive lymphocytosis. An additional point of interest in the case was the circumstance that an accident had immediately preceded the first symptoms of the disease which were observed. It is also worthy of note that the patient's father was an alcoholic subject, and was drinking heavily about the time at which conception took place.

---

## Abstracts

### PHYSIOLOGY.

**THE FUNCTIONS OF THE CAUDATE NUCLEUS.** (*Le funzioni (452) del nucleo caudato.*) G. PAGANO, *Riv. di Patol. nervosa e mentale*, July 1906, p. 289.

IN this paper Pagano gives an account of experimental researches on the functions of certain sub-cortical and medullary centres, by his own method of local injections of curara into their substance. Previous observers, in order to study the functions of the sub-cortical nuclei, have hitherto been compelled to remove the superjacent cortex, thereby introducing grave sources of error. Since stimulation of mutilated brains is bound to lead to incomplete and often erroneous conclusions, Pagano operated on un-mutilated animals, setting them at liberty immediately after the injection of the stimulating substance into the basal ganglia. By such local stimulation, the normal relations of the parts being undisturbed, it is possible to observe the effects of local exaggeration of function. The animals he employed were dogs. No anaesthetic or narcotic was employed. A flap being reflected from the scalp, a trephine-hole was rapidly made in the skull in the region of the post-crucial sulcus of the cerebrum. Then, with a fine needle, a few drops of a 2% solution of curara were gently injected, mixed with a small quantity of thionin-blue, in order that the exact site of injection might afterwards be verified anatomically. There was no tendency for the fluid to leak through the injection track if care was taken to wait a few seconds before withdrawing the needle. The sutures of the scalp wound were rapidly tied and the animal was at once set at liberty. Pagano gives full protocols of seven such experiments, and his main conclusions are as follows:—

1. Excitation of the anterior third (except its extreme tip), and of the middle third of the head of the caudate nucleus, especially in the inner half, produces in dogs an emotional picture having all the characters of *fear*, the posture of the body, the play of the facial muscles, the cardiac, respiratory, intestinal, and vesical signs, the state of the pupils, the influence of threatening gestures and especially of sounds, all producing a striking picture.

2. Excitation of the same points, but especially of the middle third of the head of the caudate nucleus, produces *priapism*, occurring immediately after the injection and sometimes persisting till the animal's death several hours later.

3. Excitation of the extreme anterior end of the caudate nucleus produces a picture of psycho-motor agitation which preserves all the characters of fear but in addition shows the phenomena of *anger*.

4. Excitation of the posterior third of the caudate nucleus produces a complex of phenomena which correspond to those of anger, especially evident in the facial expression.

5. Excitation of the outer part of the anterior third of the caudate nucleus produces, besides emotional phenomena, intestinal and vesical phenomena more marked than those produced from excitation of other parts of the nucleus.

The above results would tend to show that the caudate nucleus has a higher physiological significance than has hitherto been suspected. Pagano emphatically states that the foregoing phenomena only occur when the irritant is applied directly to the caudate nucleus itself. If the nucleus be not reached, and the injection falls on surrounding parts, the above-described phenomena are not produced. Moreover, if the motor cortex be extirpated and time be allowed for degeneration of the pyramidal fibres to occur, excitation of the caudate nucleus still produces the same phenomena. He therefore holds that there can be no question of indirect excitation through the cortex.

Nor can the phenomena be ascribed to diffusion of the fluid from the caudate nucleus into the lateral or third ventricle. In several instances he made the injection through the temporal lobe, whereby the needle avoided altogether the region of the ventricle, yet the same emotional phenomena resulted. Further, he injected the fluid in several instances into the lateral ventricle without injuring the caudate nucleus, but never produced either the emotional phenomena or the priapism. On the contrary, there were spastic tremors of the limbs with cardiac and respiratory phenomena, causing death in about an hour and a half from respiratory exhaustion. Pagano maintains that the emotional phenomena and the priapism are directly due to stimulation of the caudate nucleus, and that there exist in this nucleus centres for the expression of emotions, and also for the innervation of those vegetative organs whose activity generally accompanies emotions. He recalls the observations of other workers, notably those of Ferrier and of Bechterew, who obtained emotional phenomena by excitation of the optic thalami and corpora quadrigemina. Bechterew in particular considers the optic thalamus a reflex centre for emotional expression. Pagano has also experimented on the optic thalamus by his own method of curara injections, and quotes protocols of two such observations, showing that the emotional phenomena differed entirely from those resulting from lesions of the caudate nucleus; there were no phenomena of fear or of anger, the dog growled and howled in a plaintive fashion, and epileptiform attacks

soon appeared, increasing in frequency until the animal's death. Pagano considers that the optic thalamus is closely associated with the visual function. Finally he directs special attention to the constancy with which erection of the penis is produced by excitation of a certain limited part of the caudate nucleus.

PURVES STEWART.

**THE SUBDIVISION OF THE REPRESENTATION OF CUTANEOUS  
(453) AND MUSCULAR SENSIBILITY AND OF STEREOGNOSIS  
IN THE CEREBRAL CORTEX.** MILLS and WEISENBURG,  
*Journ. of Nerv. and Ment. Dis.*, Oct. 1906, p. 617.

THE main object of this paper is to present the following propositions : (1) that the cortical representation of cutaneous and muscular sensibility is independent of motor representation ; that it surrounds the motor zone ; and that it is subdivided into a mosaic of centres, each centre or group of centres being anatomically and functionally correlated to a motor centre or centres ; (2) that every muscle or group of muscles producing a movement or movements which are represented by separate centres in the cortex is topographically related to a segment of the skin which has also a definite cortical centre, this centre being correlated anatomically and functionally with the motor centre ; (3) that stereognostic representation, like that of cutaneous and muscular sensibility and of movements, has also its independent cortical area, and is subdivided after the manner of the motor and sensory areas. In other words, the areas of representation of movements and of sensibility and of stereognosis are separate, and are subdivided into sub-areas and centres.

Evidence for these views is chiefly clinical and pathological. Apart from many well-known instances of motor and sensory subdivisional localisation, the authors direct attention to cases (nine are briefly abstracted) where there were limitations of anæsthesia and astereognosis not only to the upper extremity, but to limited portions of this limb, and especially to the hand and certain of the fingers. The impairments of sensation present were in several instances more or less dissociated, certain forms of impairment being present, and others not, or different forms showing themselves in different parts. It is interesting to note that in four of the nine cases the impairment and disturbance of sensation were greater in the fingers towards the ulnar side of the hand, this showing itself sometimes in two and sometimes in three fingers. The authors add four more cases, studied in detail by themselves. These were all cases of cortical disease, and the facts were elicited that the touch and pain senses were more disturbed distally than proximally in the fingers and hand ; that they were more disturbed

(three cases) on the dorsal than on the palmar surface, and that astereognosis (three cases) was more persistent and decided in the middle, ring, and small fingers, and the ulnar portions of the hand. In one case, when the hand and forearm were totally relaxed, the patient had not the sensation of position and movement when the fingers were extended, although he recognised position and movement when the fingers were flexed.

Reference is made to a paper by Russel and Horsley on the representation in the cerebral cortex of the type of sensory representation as it exists in the spinal cord (*Brain*, April 1906), and their view that astereognosis is represented all over the motor cortex is combated.

S. A. K. WILSON.

**ON THE REPRODUCTION OF NERVE CELLS.** (*Sur la reproduction (454) des cellules nerveuses.*) CARMELO CIACCIO, *Rev. Neurol.*, Oct. 15, 1906, p. 876.

THIS question of reproduction of nerve cells is a much debated one, but most authors hold that once beyond the neuroblast stage, there is no reproduction on the part of the nerve cells. After lesions, however, karyokinesis has been noted in various nerve cells, but it seems never to go on to complete cell division, and may very probably only be a degeneration of the nucleus.

The author has previously recorded formation of new cells in the sympathetic system, and in the present paper records a confirmation of his results in the examination of cell reproduction in the brain of the mouse.

The process he describes is as follows:—Certain cells which occur chiefly in the outermost and innermost layers of the cortex, with large oval nuclei and very little protoplasm, are embryonic in type, and their rôle is to form new nerve cells. Unsymmetrical amitotic division of the nucleus occurs; complete splitting of the cell into daughter cells rarely occurs; but one of the secondary nuclei becomes the nucleus of a future nerve cell, while the others undergo degenerative processes, and form the cytoplasm. Exactly how they form Nissl granules and neurofibrils has not been demonstrated, but some become hypochromatic and others hyperchromatic.

He holds that he has demonstrated nerve-cell reproduction in the adult in a manner analogous to that described by Fragnito and others in the embryo. Cajal discredits this plurinuclear origin of nerve cells, saying the secondary nuclei are vacuoles wrongly interpreted, but the author thinks errors of interpretation are much more likely to be made with the reduced silver method than with staining by hæmatein and nuclear stains.

J. H. HARVEY PIRIE.

**THE MOVEMENT OF BIRDS AFTER SECTION OF THE POSTERIOR SPINAL ROOTS.** (Über die Bewegung der Vögel nach Durchschneidung hinterer Rückenmarkswurzeln.) TRENDLENBURG, *Archiv. f. Anatomie u. Physiologie*, Phys. Abteilung, 1906, H. 1, 2, p. 1.

AFTER unilateral section of the posterior nerve roots coming from the wing, no difference is to be observed in the position in which the wings are held when the bird is standing or walking. The wing reflexes which are called into action when the position of the bird is suddenly changed are brisker on the side of the operation, and wider in range at the beginning; and they can be elicited by stimuli which are not followed by any response on the normal side. Resistance to passive spreading of the wing is absent. Flying is possible, and if the bird is let fall, it uses both wings to correct itself.

After bilateral section of the same roots the position of the wings when at rest is still normal. No attempt is made to correct unusual attitudes into which they are passively put. Rising from a sitting or lying position is difficult, and the bird is quite unable to fly. If it is allowed to fall there is no attempt on the part of the wings to save it, nor if it is held up by the wings or the tail.

The condition of the wing reflexes indicates that section of the posterior roots removes a normally present inhibitory action in the wing area. When the section is unilateral, bilateral movements of the wings remain normal.

If the posterior roots from one leg are divided the bird cannot walk or stand at first, but after a time it learns to walk, although on the operated side the leg is lifted too high, the stride is lengthened, and the rhythm of gait is therefore altered. If the section is bilateral the bird is quite unable to stand, and sits with its feet spread out in front.

Evidently after bilateral section of the posterior roots from the limbs, sensory stimuli do not reach higher centres, and after unilateral section there is no compensatory innervation from the opposite side, as in the case of the wings. The overaction of the affected leg is due to the removal of normal reflex inhibitory influences exercised on muscular action *via* the posterior roots, as well as *via* central paths.

If, in addition to the operation on leg roots, the labyrinth is unilaterally or bilaterally extirpated, compensation becomes impossible. Vision exercises no influence over the resulting ataxia. Apparently the condition of tone of the wings does not depend on cerebral influence, for after removal of the cerebrum no alteration is observable; further, the decerebrate pigeon can attempt to execute movements of compensation when its posterior roots from the legs have been divided.

S. A. K. WILSON.



**PSYCHOLOGY.**

**THE SCIENTIFIC INVESTIGATION OF THE PSYCHICAL FACULTIES OR PROCESSES IN THE HIGHER ANIMALS.** By IVAN PETROVITCH PAWLOW, M.D., Professor of Physiology, University of St Petersburg, *Lancet*, October 6, 1906, p. 911.

IN the Huxley Lecture delivered at Charing Cross Hospital on October 1st, Professor Pawlow gives a summary of the conclusions already published with respect to "conditioned" reflexes, and an account of work in the same direction recently done in his laboratory. The subjects of the experiments were dogs in normal condition, and the reflex specially studied was the action of the salivary glands.

A reflex action is a specific response invariably given to a specific stimulus; a "conditioned" reflex is a specific response which sometimes, but not invariably, follows a specific stimulus. The problem is to discover the laws which determine the response or failure of response in the case of a conditioned reflex. To illustrate: when food is placed in the mouth of a dog the excretion of saliva invariably follows—this is a reflex action; when food is shown to a dog, the excretion of saliva sometimes takes place and sometimes does not—this is a conditioned reflex.

These two classes of action manifestly resemble one another closely; in both we have primary stimulation of the centripetal paths and secondary stimulation of the centrifugal paths, with in both cases the intervention of the central nervous system. The difference lies in the mode of this intervention, and this difference has up till now been considered so great as to justify the assignation of only the one set of actions to physiology, while the other is reserved for psychology. Professor Pawlow's endeavour is to break down this division-wall and to restore to physiology what, in his opinion, properly belongs to her.

Certain facts which have been ascertained with regard to these conditioned reflexes have been already published. The chief of these are:—Every conditioned stimulus becomes ineffective on repetition, and the shorter the interval between the repetitions the more quickly is the reflex obliterated. Spontaneous restoration takes place only after the lapse of one or two or more hours. If a conditioned stimulus is employed for a somewhat long time—days or weeks successively—it loses its power altogether; for example, if a certain kind of food is shown to a dog without being given him to eat, his salivary glands in time cease to become active at the sight. Any conditioned stimulus which has been rendered im-

tent may be restored at once by being made to act along with the unconditioned stimulus.

From these facts it was natural to suppose that the conditioned reflexes arose from their stimuli being constantly associated with the stimuli producing unconditioned reflexes. The next step obviously was to try whether conditioned reflexes could be manufactured. This attempt was crowned with success. It was found that any stimulus whatever—such as heat, cold, or rubbing applied to the skin, the ringing of a bell, the sight of an electric light—could be transformed into a stimulus bringing about excretion from the salivary glands. The method was simply to let these stimuli act invariably for a certain number of times along with the unconditioned stimulus—the placing of something in the dog's mouth. The artificial reflexes thus manufactured showed exactly the same characteristics as the natural ones.

It thus appears that stimuli which always synchronise with stimuli which give notice of events of importance to the organism become themselves "signalling stimuli," and themselves bring about the necessary adjustment; and the fact that they cease to act when they no longer regularly accompany the unconditioned stimulus is further evidence of the marvellous delicacy of reaction which characterises the organism in its relation to its environment.

The time required to establish conditioned reflexes was next investigated, and it was found that this bore some relation to the strength of the stimulus. Thus a temperature of 0° or 1° C. would give a reaction by itself after it had been made to accompany the unconditioned reflex twenty or thirty times; whereas a temperature of 4° or 5° C. gave no reaction after a hundred repetitions. The same thing happened with respect to 50° and 45° C. respectively.

The attempt was next made to find out the elements of a stimulus, or, in other words, to discover what amount of differentiation the nervous system of a dog is capable of. It was found that if the application of cold to a definite area of the skin has been made to act as a conditioned stimulus, then the application of cold to another region of the skin causes secretion of saliva on the very first occasion. Hence the stimulus of cold generalises itself over a considerable portion, perhaps even over the whole, of the skin. The same is true of heat, but not of mechanical stimulation. Great delicacy of analysis is manifested with respect to musical sounds; notes which differ by as little as a quarter of a tone from the note which has been established as a conditioned stimulus will sometimes fail to give a reaction.

Experiments were also framed with a view to testing for traces or latent remnants of both conditioned and unconditioned stimuli.

Thus a conditioned stimulus was allowed to act one minute or even two minutes before the application of the conditioned stimulus. Conversely, it was not brought into action until the unconditioned reflex was at an end. In all these cases the artificial reflex was developed. Indeed, in the case where the conditioned stimulus acted first, and was separated from the unconditioned one by an interval of two minutes, an unusually copious excretion of saliva took place.

The far-reaching importance of these discoveries is obvious. The bringing of these conditioned reflexes into the domain of law is undoubtedly a definite step towards that mechanisation of the central nervous system which every physiologist, as such, is bound to presuppose. Professor Pawlow rejoices openly in the fact that by these researches he removes a whole field of investigation from the baneful influence of psychology. The answers to the questions which he propounds to nature are delivered objectively ; no transference of feelings distinctively human to the animal is possible ; the results are all amenable to the laws of quantity, and the method is thus evidently in accord with the demands of the most exact science.

The tendency of the investigation is summed up in the following remarkable sentence :—

“ Men will possess incalculable advantages and an extraordinary power over themselves when scientific investigators will subject other men to the same external analysis as they would employ for any natural object, and when the human mind will contemplate itself, not from within, but from the outside.”

We may, however, perhaps be allowed to remark that in this contemplation from the outside the whole meaning of the drama of human life is missed. The play of atoms and molecules in the human brain is a purely hypothetical construction of science ; the whole reality of the universe is in the inner world of emotion, cognition, and conation. Nevertheless every true psychologist will be grateful for the light which these studies of Professor Pawlow throw on the outskirts of his subject, and when the further results which the Professor foresees are given to the world, he will be prepared to work them into correlation with his own conclusions derived from a direct study of the laws of consciousness.

MARGARET DRUMMOND.

**PATHOLOGY.**

**COLLATERAL REGENERATION BY NERVE FIBRILS WITH  
(457) CLUB-LIKE TERMINALS IN PATHOLOGICAL AND  
NORMAL CONDITIONS; TABETIC LESIONS OF SPINAL  
ROOTS.** (*Régénération collatérale de fibres nerveuses  
terminées par des massues de croissance, à l'état pathologique  
et à l'état normal; lésions tabétiques des racines médullaires.*)  
J. NAGEOTTE, *Nouv. Icon. de la Salpêtrière*, No. 3, 1906, p. 217.

THE author draws attention to two special changes which can be demonstrated by Ramon y Cajal's new ammonia-alcohol and reduced silver method, in the root fibres and in the ganglia of the posterior roots and their processes in the course of tabes. (1) In tabes there is moniliform swelling of the axis-cylinders. This change affects the fibres of the posterior roots from the bifurcation of the axone near the ganglia as far inwards as the posterior columns. It involves the large and medium-sized fibres, but spares certain very fine fibres which probably pre-exist in the root and become more evident owing to the degeneration of the other larger fibres. In the anterior roots this moniliform swelling is found at the level of and below the foci of interstitial neuritis which Nageotte has described in tabes, but not between this and the cord. (2) The author has demonstrated that in tabes the cells of the spinal ganglia and their axones give origin to fine fibrillæ. These fibrillæ are exceedingly numerous in advanced tabes, and they are evidently compensatory to the destruction of old root fibres. They rise either from the cell body or from the portion of its axone contained within the peri-cellular capsule, or lastly from the extra-capsular portion of the nerve fibres. Those which arise from the cell body frequently remain included by the peri-cellular capsule. Those which arise from the axis-cylinder processes of the cells bud off in the manner of collaterals. In advanced tabes these new-formed fibrils frequently ramify greatly. The fibrils which arise from the cell body and from the fibrils within the capsule for a time ramify within its capsule. Eventually they may perforate this, and with the club-like processes in which they terminate, they tend to pass from the ganglion towards the nerve root. In their passage they undergo numerous tortuosities and ramifications. They all tend to agree in the presence of rounded or oval, more or less regular balls, but their most remarkable characteristic is the presence of a terminal club-like mass contained in a nucleated capsule. These appear to represent the cones of growth of the embryonic

period of development. The masses vary considerably in shape. They never show the presence of neuro-fibrillæ. They all have a peculiar tendency to be directed towards the cord. In incipient tabes they accumulate at the superior pole of the ganglion. Others pass into the posterior root, but none of them succeed in crossing any focus of interstitial neuritis, and none have been traced so far as the posterior columns.

In the grey matter of the cord Nageotte has also traced somewhat similar club-shaped processes along the anterior margin of the anterior cornu, and also along the internal margin of the posterior cornu. The clubs in this instance, however, differ from those of the fibrils arising from the posterior root ganglia in the absence of a nucleated capsule.

Nageotte is of opinion that these fibres terminated in clubs are due to regeneration for the purpose of replacing destroyed root fibres. He does not think it probable that the regeneration is sufficient to restore lost function. He employs the term collateral regeneration because the new fibres develop after the manner of collaterals from parts of the neurone nearest the vital centre, and to distinguish the process from that of *terminal* regeneration, which is found in divided nerves. It is probably not limited to tabes, and is merely an exaggeration of processes which go on in healthy nervous systems.

The paper is illustrated by a series of beautiful illustrations of the author's microscopical preparations.

ALEXANDER BRUCE.

**EXPERIMENTAL CEREBRO-SPINAL MENINGITIS AND ITS  
(458) SERUM TREATMENT. SIMON FLEXNER, *Brit. Med. Journ.*,  
Oct. 20, 1906, p. 1023.**

As tested upon laboratory animals, the *Diplococcus intracellularis* of Weichselbaum is a micro-organism of low and variable pathogenicity. Freshly isolated cultures are usually pathogenic for small guinea-pigs, whereas cultures grown for a period on artificial media are not. Active cultures injected into the peritoneal cavity of guinea-pigs often cause death in eight to ten hours; the animals may, however, die in four hours or survive thirty-six hours.

In cases of rapid death from virulent cocci the exudate shows large, often prodigious, numbers of cocci. The appearances are indicative not only of preservation of the injected cocci, but are suggestive of their multiplication. The virulence of the cocci is proportionate to their resistance to destruction. A relatively non-virulent strain of the coccus may still be toxic, but has little

or no capacity to resist disintegration, and none for multiplication in the peritoneum. The peritoneal exudate, after having been freed from cells by centrifugalisation, still possesses a high digestive power for cocci, and the exudate, after having been heated to 58° C. for 30 minutes, has the same digestive power as before on fresh cultures, and on those heated to 65° C. It therefore appears that although a measure of the reaction of the guinea-pig is found in the emigration of leucocytes, the disappearance of cocci from the peritoneal cavity does not depend wholly upon phagocytosis, for it seems probable that the removal of the cocci may be effected by their self-digestion and by the digestive action of the inflammatory exudate.

In monkeys, intraspinal injection of cultures produces either an acutely fatal leptomeningitis or an acute disease from which recovery usually takes place in three or four days. Monkeys which survive beyond the second day after inoculation tend rather to recover than to die. The early disappearance of the cocci from the spinal canal, early emigration of leucocytes, active phagocytosis and dissolution of the cocci both within and without leucocytes are favourable signs; yet the monkey may succumb although cocci cannot be found in smears nor obtained in cultures from the inflamed membranes.

A large dose of an active culture is required to cause marked symptoms in monkeys or to bring about their death from meningitis, and the author believes that in many of the experiments no multiplication of the cocci took place.

In monkeys the inflammatory exudate was found chiefly in the spinal meninges and the meninges at the base of the brain, but the inflammation was also found to extend into the ethmoidal sinuses and probably into the nose. Attempts to isolate the cocci from the nose were, however, unattended with success.

Antiserums were prepared in rabbits, goats, and large monkeys. The goat proved more satisfactory than the rabbit, and eventually yielded a serum of marked agglutinative power. Although this serum, when given intraperitoneally, was capable of protecting guinea-pigs against the diplococcus, its intraperitoneal injection failed to exert any beneficial effect upon the monkey. The antiserums made in large monkeys (*Macacus nemestrinus*) were used on a series of five smaller monkeys (*Macacus rhesus*) which had been injected with fatal quantities of the diplococcus. Whereas the control monkeys all died within twenty-four hours, of those which received the antiserum four either remained well or recovered from the disease, and one died nineteen hours after infection.

In how far the results obtained with guinea-pigs and monkeys can be applied to the prevention and treatment of cerebro-spinal

meningitis in man it is not safe to predict, but experiments dealing with these important questions are being undertaken.

W. T. RITCHIE.

**COMPARATIVE STUDIES ON THE LOCAL ACTION OF COCAINE  
(459) AND STOVAIN ON PERIPHERAL NERVES.** SANTESSON,  
*Festschrift für Olaf Hammarsten, xv.*

THE author records the results of a number of experiments undertaken to test the relative powers of cocaine and stovaine on peripheral nerves, motor and sensory. Frogs and rabbits were used in the investigation, and contradictory results, of which no satisfactory explanation could be given, were obtained. The results in rabbits approximated most closely to the well-known effects of cocaine and stovaine in man. Microscopic examination of the treated nerves seemed to show alterations dependent on the action of the drugs, but other causes, such as the handling of the nerves, might have produced the changes, which were in any case minimal and almost without significance. The whole investigation appears to have been devoid of positive results and to have added nothing to our knowledge of the action of cocaine or stovaine.

J. W. STRUTHERS.

**CLINICAL NEUROLOGY.**

**ASCENDING NEURITIS AND CHRONIC RHEUMATISM (Névrite  
(460) ascendante et rhumatisme chronique.)** P. LEJONNE and  
M. CHARTIER, *Rev. Neurolog.*, Oct. 15, 1906.

THIS is the account of a case in which an ascending neuritis of the nerves of the left arm and chronic rheumatism of the metacarpophalangeal and inter-phalangeal joints of the same side, followed on an injury to the terminal phalanx of the middle finger. The first symptom was excessively severe pain in the fingers, later spreading to the hand, forearm, and upper arm. Gradually there followed loss of function and muscular wasting of the hand and arm. There were no sensory changes and no alterations in the electrical reactions. The nerves were thickened and tender to pressure. The symptoms of neuritis preceded the appearance of the arthritic changes, and only those joints supplied by the affected nerves were involved. The joints and periarticular tissues were probably predisposed to the onset of the infective rheumatic process by the loss of trophic influence resulting from the nerve changes.

HENRY DUNBAR.

**ON THE CLINICAL ASPECTS OF THE NEURAL FORM OF  
(461) PROGRESSIVE MUSCULAR ATROPHY. (Zur Klinik der  
neuralen Form der progressiven Muskelatrophie.) STIEFLER,  
*Zeitschr. f. Heilk.*, 1906, H. 8.**

THIS paper forms a contribution to the literature of that type of chronic muscular atrophy described first by Charcot and Marie, as well as by Tooth, and known as the "neural" or "peroneal" type.

The writer, as the result of thorough investigations into the subject, discovered 240 cases. Of these 145 occurred on a hereditary basis, 44 in families of which other members were affected, while 47 were isolated cases. Thus three-fifths are hereditary, one-fifth of family occurrence, while one-fifth are sporadic. The series of cases recorded in this paper (numbering 19) were traced through four generations and were transmitted, like the cases of other writers, through both men and women. In this series the proportion of men affected was three to every one woman; while other writers give an even higher disproportion.

The usual time of onset the writer found to be in the second decade of life, and the disease makes but little progress after the age of 40, while many of the affected persons reach the age of 60 or 70 years. All the cases, save one, showed their first symptoms in the lower limbs; while the exceptional case, in which the wasting was first seen in the hands, was further atypical in that it commenced at the age of 68. In two brothers there were extensive changes in the bones as well as in the muscles, and the symptoms of one of these cases are given in full detail.

The paper is illustrated by three plates, and there is a copious bibliography.

JOHN D. COMRIE.

**A CASE OF LANDRY'S PARALYSIS, WITH RECOVERY.  
(462) WHARTON SINKLER, *Journ. of Nerv. and Ment. Dis.*, Nov. 1906,  
p. 692.**

DR SINKLER reports a case of Landry's paralysis, with recovery, in which the bulbar nerves were involved.

He discusses briefly the diagnosis of the disease from multiple neuritis, poliomyelitis, and spinal paralysis, and notes the different views held on its pathology, and the probability of microbic origin.

His patient was a healthy man, aged thirty. On the first day of illness he felt unaccountably tired, and had prickling in the fingers.



Second day, stiffness, difficulty in walking. Fifth day, pain in calves, no tenderness, weakness in arms and legs. Eighth day, could walk only with strong support; numbness in hands and feet, no objective sensory change; pupil and epigastric reflexes present, abdominal and all deep reflexes abolished. Tenth day, almost complete paralysis of arms and legs, weakness of lower face, deglutition unaffected. Twelfth day, weakness in upper face, difficulty in deglutition. Electrical reactions of muscles normal. Fourteenth day, slight improvement in face. Twentieth day, improvement in deglutition and power of arms.

The improvement was maintained, and the legs regained power. Two weeks later he sat up, and in six weeks could walk. Three months later he was in perfect health, but the deep reflexes were still absent.

Two years later he was still in good health, and the knee-jerks had returned.

D. W. CARMALT JONES.

**CONTRIBUTION OF A CASE TO PAL'S TEACHING AS TO THE  
(463) VASCULAR CRISES OF TABELIOS. (Ein kasuistischer  
Beitrag zu Pals Lehre von Gefässkrisen bei Tabiker.) MÖRCHEN,  
*Neurolog. Centralbl.*, Oct. 16, 1906, p. 940.**

THE writer records with great prolixity the history and symptoms of a case of tabes, with a development at a late stage in the disease of cerebral disturbances. The patient became at times extremely exhausted and sleepless, and on several occasions had attacks in which he lost consciousness, and had great irregularity of the heart's action, and congestion of the face and head. These symptoms were found to be much alleviated by doses of digitalis.

According to Pal's investigations the blood-pressure varies much in tabetics—falling, for example, during attacks of lightning pain, and rising in gastric crises; while in high-pressure crises epileptiform conditions may ensue. Pal further found that physostigmine, belladonna, and iodine, as well as reflex agencies like hot hand-baths, served to diminish the tension and relieve this condition. The present writer believes that the symptoms in his case were of similar origin, and recommends the use of digitalis, which benefited it.

JOHN D. COMRIE.

**POLIOMYELITIS ANTERIOR ACUTA AND MENINGITIS CERE-  
(464) BROSPINALIS. TIEDEMANN, *Muench. Med. Wochenschr.*,  
Oct. 23, 1906, p. 2095.**

THE writer discusses at some length with references to recent literature the relationship existing between encephalitis of the

grey matter, acute poliomyelitis of the cord, and meningitis. He quotes researches of Strümpell and of Medin, for example, which show that encephalitis and poliomyelitis may be due to the same infective process, the latter authority having observed in an epidemic among children that in some cases the cranial and in others the spinal nerves were affected. Other observers, as Dauber and Schultze, are quoted as proving that meningitis may accompany poliomyelitis acuta; one such combined case of the last-named writer showed the Weichselbaum-Jaeger bacillus in the fluid drawn by lumbar puncture.

The writer gives full details of an illustrative case, in which a factory girl of 17, after a febrile attack lasting some days, and diagnosed as "influenza," became lethargic, suffered from pain in head, neck, and spine, had optic neuritis and extensive paralysis of the right upper arm and forearm with R.D., and presented in her cerebro-spinal fluid a turbidity with great lymphocytosis and leucocytosis. The patient ultimately recovered, though several groups of arm muscles remained paralysed and atrophied. The diagnosis of acute poliomyelitis accompanied by meningitis was made, and the writer discusses the various features of the case at length.

JOHN D. COMRIE.

**A CASE OF WESTPHAL'S PSEUDOSCLEROSIS.** SIMPSON, N.Y.  
(465) *Med. Journ.*, Sept. 29, 1906, p. 645.

THE writer simply gives the clinical features of the case of a girl, aged 21, in whom he diagnosed this condition. The chief points noted were—Hereditary psychoses; weakness and loss of memory gradually setting in; slow speech; ataxia, shown by the hands and in the gait; headaches; increase of the knee-jerks; hypæsthesia, especially on the left side. The patient still lives, so that the pathological state is not recorded.

JOHN D. COMRIE.

**THE DIFFERENTIAL DIAGNOSIS BETWEEN MULTIPLE  
(466) SCLEROSIS AND THE PSYCHOGENIC NEUROSES.** (Zur  
Differentialdiagnose zwischen psychogener Neurose und  
multipler Sklerose.) HELLER, *Klinik. f. psych. u. nerv. Krank-*  
*heiten*, Bd. 1, H. 3, 1906.

THIS paper is intended to demonstrate the value of the technical methods employed by Dr Sommer, and described in his "Diagnostik der Geisteskrankheiten," in the differential diagnosis between organic and functional nervous disease in doubtful cases, and takes the

form of a careful study of a particular case. The case does not appear to have been, however, one admitting of much doubt, as on admission to the Klinik there were present, in addition to a certain amount of mental reduction, and mentioning only the more prominent symptoms, the following signs:—increased knee-jerks, ankle-clonus, spastic gait, staccato monotonous speech, myasthenia, intention-tremor, and optic atrophy. The main interest of the paper lies in the author's analysis of the curves, recorded by Sommer's instruments, of the knee-phenomena and finger tremor.

R. CUNYNGHAM BROWN.

**SYRINGOMYELIA, EXTENDING FROM THE SACRAL REGION (467) OF THE SPINAL CORD THROUGH THE MEDULLA OBLONGATA, RIGHT SIDE OF THE PONS, AND RIGHT CEREBRAL PEDUNCLE TO THE UPPER PART OF THE RIGHT INTERNAL CAPSULE (syringo-bulbia).** WILLIAM G. SPILLER, *Brit. Med. Journ.*, Oct. 20th, 1906, p. 1017.

NOTES, clinical and pathological, with illustrations, are given of a case of syringomyelia whose special interest lies in the syringo-bulbia and the vertical extent of the lesion. The cavity, beginning in the lower sacral region, implicates the left posterior horn in the sacral and lumbar regions: in the thoracic and cervical regions, both posterior horns and the central grey matter are involved, the cord being very distorted above the mid-thoracic region. The lower part of the medulla-oblongata shows a transverse cavity in each anterior pyramid; the fibres of the right hypoglossus nerve are completely cut across, thus explaining intense atrophy of the right side of the tongue; higher up in the medulla the cavity in the left anterior pyramid has disappeared. In the pons the cavity is transverse in the right pyramidal tract; there is partial degeneration of the right abducens nerve from implication in the cavity, thus explaining paresis of the right abducens nerve. In the right cerebral peduncle, the cavity is in the substantia nigra, extending into the upper part of the crura. In the lower part of the right internal capsule the cavity becomes divided, one part remaining nearer to what will become the knee of the capsule, the other occupying the posterior part of the capsule and soon disappearing. The former, at a higher level, occupies the inner segment of the globus pallidus and outer portion of the internal capsule near the knee. Still higher, there is a cavity on each side of the knee, one in the posterior part of the head of the caudate nucleus, and the other in the anterior part of the lenticular nucleus, connected by a narrow slit, the cavity in the head of the caudate nucleus extends

to within 2 or 3 mm. of the lower surface of the lateral ventricle, and does not open into the ventricle.

Schlesinger finds that the cavity in syringomyelia has never been observed above the upper end of the facial nucleus, so that only the fifth to the twelfth cranial nerves are ever implicated in syringobulbia, while the involvement of the higher cranial nerves is caused by complications (*e.g.*, hydrocephalus, tabes, etc.). Spiller's case shows that these higher nerves might be involved by the cavity, and that their implication does not necessarily imply a complicating disorder, although he agrees with Schlesinger that, in the majority of cases, symptoms in the supply of the upper cranial nerves point to a complicating lesion. Hoffman's opinion, that invasion of the pons in syringomyelia is not more frequently seen because vital centres are implicated before the cavity extends to the pons and thereby death is caused, is shown by Spiller's case to be untenable, as a cavity may extend a long distance in the medulla and pons without impairing many cranial nerves.

A. W. MACKINTOSH.

**ON TRANSITORY HEMIPLEGIA IN ELDERLY PERSONS. F. H. (468) EDGEWORTH, *Scot. Med. and Surg. Journ.*, November 1906.**

THIS condition is probably due to a temporary spasm of a branch of one of the cerebral arteries. It can usually be distinguished from hemiplegia, the result of an organic lesion, by the absence of the extensor plantar response. The writer records two cases, both in males—one at 68, the other at 64 years of age. In both clonic spasm of the affected side preceded the paralysis. The first patient was subject to temporary mental derangement, and the second had attacks of clonic spasm not followed by paralysis.

HENRY DUNBAR.

**TWO CASES ILLUSTRATING POINTS IN THE DIAGNOSIS OF (469) TUMOUR OR OTHER LESION OF THE UNCINATE REGION OF THE TEMPORO-SPHENOIDAL LOBE. THOMAS BUZZARD, *Lancet*, July 1906.**

In this lecture the author records two cases which illustrated the chief points in the diagnosis of tumour of the uncinatè region of the temporo-sphenoidal lobe. Special attention is drawn to the presence of "intellectual auræ or warnings" in lesions of the

uncinate region, a condition to which Hughlings Jackson gave the name of "the dreamy state."

Both cases were very similar in their clinical features, but the situation and nature of the lesion was determined only in the second case which came to necropsy. The second case was that of a woman aged 21, who was first seen in November 1904. Her family history and previous health were unimportant. She had suffered from attacks of vertigo for two years, and four months before admission she began to complain of severe attacks of headache followed later by vomiting and attacks of "loss of mind," which were typical examples of the "dreamy state"; she also suffered at times from a dull aching numbing pain in the right arm and leg, which would last for a few minutes, and on occasions she had a similar sensation on the left side. Although the patient often woke up with a bad taste in her mouth which persisted all day, there was no taste sensation directly associated with the psychical attacks.

The examination of the nervous system revealed no organic signs, and the subjective symptoms passed away while she was in hospital. A diagnosis of right temporo-sphenoidal tumour was made.

In November 1905 (one year later) she was re-admitted to hospital. She had had no more "dreamy states," but had had "feelings of dread," accompanied by a cold, creepy sensation all over the body. The headache had returned, and her sight was affected. At times she had attacks, characterised by feelings of contraction associated with coldness and numbness, generally on the right side, but sometimes on the left. On examination, there was optic neuritis, slight weakness of the left side, with a tendency to lurch to the left when she walked.

Taste and smell were not lost, and there was no affection of sensation. The superficial abdominal reflexes were absent on the left side, otherwise the reflexes were normal.

The patient died suddenly in bed.

The necropsy revealed a glioma situated in the right hippocampal gyrus, which was enlarged; this destroyed the uncus and extended into the inferior horn of the lateral ventricle; its anterior end infiltrated the lenticular nucleus, but did not destroy the internal capsule. The right optic tract and the right crus cerebri, were compressed and flattened by the growth.

In discussing the cases, attention is called to the "dreamy states," the "taste aura" present in the first case, and to the loss of the superficial abdominal reflexes on the side opposite to the tumour without any alteration of the corresponding plantar reflex.

T. GRAINGER STEWART.

**LIMITED AREA OF ANÆSTHESIA, EPILEPTIFORM ATTACKS  
(470) OF HEMIALGESIA, AND EARLY MUSCULAR ATROPHY  
IN A CASE OF BRAIN TUMOUR. MORTON PRINCE.**

Operation by JOHN C. MUNRO, *Journ. of Nerv. and Ment. Dis.*,  
Nov. 1906, p. 698.

THE case was one of cerebral tumour of the left Rolandic area, with unusual sensory symptoms.

The patient, a girl of 19, suffered from recurrent attacks of pain beginning in the fingers of the right hand, and gradually involving the right arm, right side of face and body, and right leg, and accompanied by spasm of the right arm. In the attacks there was some disturbance of consciousness, and they were regarded as epileptiform.

There was an area of slight anæsthesia involving the right face, neck, and shoulder only. There was weakness and general wasting of the muscles of the right arm, but the electrical reactions were normal. The deep reflexes were increased on the right, but there was no Babinski. There was some headache and nausea, and double optic neuritis.

The condition progressed, and she became blind, and quite hemiplegic on the right; the slight tactile anæsthesia involved the whole of the right arm, and there was complete loss of muscle sense and stereognosis in it. Babinski's sign appeared on both sides.

A diagnosis was made of cerebral tumour of the right cortical arm area, localised in the post-central convolution, and spreading forwards into the ascending frontal gyrus, and backwards into the parietal lobe.

A sub-dural endothelioma was removed from the middle of the Rolandic area, involving both central convolutions, but not spreading back. The patient died, and there was no autopsy.

The findings in this case are compatible with the theory that the post-central gyrus is sensory, and includes both tactile and muscular senses and perhaps stereognosis.

D. W. CARMALT JONES.

**SEXUAL INFANTILISM WITH OPTIC ATROPHY IN CASES OF  
(471) TUMOUR AFFECTING THE HYPOPHYSIS CEREBRI.**

HARVEY CUSHING, *Journ. of Nerv. and Ment. Dis.*, Nov. 1906,  
p. 704.

TUMOURS of the hypophysis cerebri in women often lead to amenorrhea. Axenfeld has reported certain cases—one of them in

a woman who had never menstruated, was sexually undeveloped, and whose optic discs showed a primary, not a consecutive atrophy. Dr Cushing has seen a similar case. Abelsdorff has quoted a case of presumed benign tumour of the hypophysis, in which amenorrhea preceded other symptoms by ten years.

Two new cases are here described at length.

(1) A girl of 16, who had never menstruated, had undeveloped breasts and scanty pubic hair. She had small hands and unusually tapering fingers. She complained of headache, pain in the back, malaise, nausea, vomiting, and imperfect vision. Cranial nerves and discs were normal.

Later she developed double optic neuritis, severe headache, and projectile vomiting. She became mentally dull, vision failed till she could hardly count fingers, and there was general contraction of the fields, but no bitemporal hemianopia. Craniotomy was performed and alleviated the symptoms and the oedema of the discs, but after a further operation the patient died of pneumonia.

*Post-mortem.*—A tumour containing cartilage cells was found occupying the position of the tuber cinereum, and projecting into the third ventricle. It is suggested that this tumour had existed for years and retarded sexual development by pressure on the hypophysis, and that the acute terminal symptoms and optic neuritis were due to its displacement into the third ventricle.

(2) A woman of 26, who had only once menstruated, and was sexually infantile. She had tapering fingers and some boggi-ness of subcutaneous tissue. She complained of headache, failing vision, and right trigeminal neuralgia. No vomiting. She had been blind in the left eye for four years; this may have begun as a temporal blindness. The right field of vision was irregularly contracted. Both discs were atrophic, especially the left. Smell was diminished on the right, and the right trigeminal area was partially anæsthetic. The axes of the eyes were not always parallel.

Bilateral craniectomy was performed, with relief of the headache and neuralgia and some improvement in the sight of the right eye, with some enlargement of the temporal field and appreciation of colours on the temporal side hitherto absent; there remains a nasal hemianopia for colours.

A skiagram shows some shadow with deformity about the sella turcica.

The diagnosis suggested is a benign tumour, probably congenital, causing amenorrhea by pressure on the hypophysis.

The author regards amenorrhea in a woman, associated with optic atrophy, as diagnostic of tumour pressing on the hypophysis.

D. W. CARMALT JONES.

**DIFFERENTIAL DIAGNOSIS OF CEREBRAL DISTURBANCES  
(472) OF TOXIC ORIGIN DUE TO ALCOHOL AND TOBACCO  
AND TO GENERAL PARALYSIS ACCORDING TO THE  
OCULAR SYMPTOMS.** (Diagnostic différentiel des troubles  
cérébraux, etc.) A. RODUT and F. CANS, *Annales Medico-Psychologiques*, Nov.-Dec. 1906, p. 408.

IN discussing the differential diagnosis between chronic alcoholism with delirium and general paralysis, the authors quote a statement of Magnan that "alcohol can mask, simulate, or cause general paralysis." They point out the importance of the eye symptoms in this connection, grouping these under five heads: (1) Pupillary changes; (2) alteration in sensibility; (3) aberrations, illusions, and hallucinations of vision; (4) alterations in the fundus; (5) visual troubles. In chronic alcoholism the pupils are contracted, equal in size as a rule, and they have a sluggish reaction to light; a complete Argyll-Robertson pupil is rare. Inequality of the pupils occurs sometimes during the acute attack, but is not of importance.

In general paralysis a difference in the size of the pupils is found, but is only of importance when associated with other signs. In this disease there is a progressive change in the reaction of the pupils, Argyll-Robertson, false Argyll-Robertson, *i.e.* loss of light reflex, with diminution of accommodation, and finally, complete immobility of the pupils. This change may affect the two eyes differently. Deformities and irregularities of the pupils are present in both conditions; the eccentric pupil they find commoner in general paralysis, tabes, and cerebral syphilis.

The authors then discuss the alterations in the sensitiveness of the eye. They describe a peculiar form of hemianæsthesia of the cornea in alcoholism, resembling that in hysteria, though not so complete, and differing in the fact that in the alcoholics reflex stimulation of tears can be produced by touching the anæsthetic area, which is not present in the case of hysteria. They then go on to describe the differences in the hallucinations of sight, etc., in the two conditions, and give a very full account of the changes in the fundus of the eye, and the extent to which vision is affected, and in what particular manner, in the intoxications by alcohol, tobacco, and general paralysis: and they have remarked that with atrophy apparent in the fundus there is a remarkable preservation of sight in the general paralytic, while the extent of the loss of sight in the alcoholic is not to be explained by the extent of the changes in the fundus.

DUNCAN LORIMER.



**REFLEX EPILEPSY.** (*Ueber Reflexepilepsie.*) ERNST URBAUT-(473) SCHITSCH, *Wien. klin. Wochenschr.*, Sept. 27, 1906, p. 1160.

THE main tenor of the paper is the importance of clearly separating the two conditions that are confused under this title. On the one hand we find cases of true epilepsy in which peripheral irritation bears a relation to the actual attacks, though not to the disease itself. This condition should be called Reflex Epilepsy. On the other hand there are cases in which attacks indistinguishable from those of true epilepsy occur, which are due solely to peripheral irritation. These should be called Reflex Epileptiform Attacks (*reflektorisch-epileptiforme Anfälle*). The author lays great stress on the significance of nomenclature here, as elsewhere, for even a physician who is quite aware of the facts is unconsciously influenced, if he uses the term Reflex Epilepsy for both conditions, in the direction of considering both states as epileptic; and that these are absolutely distinct conditions is insisted on throughout. The differential diagnosis is therefore most important, as the prognosis and treatment are quite different. Unfortunately, however, it is frequently extremely difficult, and several cases illustrating this difficulty are referred to. One of the main distinctions is this fact: an epileptic always has other evidence of neurosis, both in his family and his personal history, so that the attacks are only one element in the case; the epileptiform case, on the other hand, is perfectly sound mentally, and has no other sign of disease of the nervous system. The effect of operative treatment on the local condition is different in the two cases. In true epilepsy the attacks may be relieved, and may cease for a while; but, on account of the underlying predisposition, sooner or later they return: frequently the recurrence is due to some other local irritation. One does not gather, however, that the author would deprecate local treatment even in these cases. The effects of this treatment in the epileptiform cases are striking, permanent cure usually resulting. It is absurd, however, for reasons given above, to label such results "Cure of Epilepsy." Bromide medication, while favourably affecting most cases of pure epilepsy, has but little influence on the epileptiform cases. In the treatment of epilepsy the author lays stress on the importance of careful dieting. He founds this opinion both on the results of experience and on the view that the intestinal tract is frequently the source of irritation which—given a suitable predisposition—causes true epileptic attacks. He tries to correlate this gastric and intestinal irritation with the common ascending epigastric aura, but only in a far-fetched manner. The other sources of irritation are referred to at some length, and several cases described.

ERNEST JONES.

**A NOTE ON PECULIAR ATTITUDES IN EPILEPSY DURING (474) SLEEP.** N. B. Ross, *New York Med. Journal*, Oct. 6, 1906, p. 689.

ATTENTION is drawn to the fact that the low grade epileptic patients frequently assume remarkable attitudes during sleep. For instance, they may lie laterally in such a flexed position that the forehead is between the knees, and asphyxia may occur. A sitting posture during sleep is not rare. One patient had for twenty months lain in his sleep flat on his abdomen. Another slept always with his head hanging over the side of the bed in a dependent position. Photographs are given of these different positions.

ERNEST JONES.

**EPILEPSY AND MIGRAINE.** (L'épilepsie et la migraine.) P. (475) KOVALESKY, *Arch. de Neurol.*, May 1906.

THE author reports a case of epilepsy in a woman of thirty-seven, subject to migraine from the age of eight; the convulsions occurred at the end of coitus, and were preceded by an aura of a red light. He holds that in certain cases migraine and epilepsy can be combined and substituted for each other; this shows their close relation. Outbursts of fury without reason may be epileptic equivalents. He emphasises the connection of the fit with the sexual act.

C. MACFIE CAMPBELL.

**SOME OBSERVATIONS ON CONVULSIONS IN CHILDREN, AND (476) THEIR RELATION TO EPILEPSY.** R. O. MOON, *Lancet*, Sept. 15, 1906.

THE author in this paper gives analysis of a second set of 100 cases.

In his opinion "convulsions in early life may shade off indefinitely into epilepsy or epileptiform manifestations, so that it becomes often impossible to say where the one stops and the other begins."

In about 50 per cent. of cases the first convulsion arose without any obvious cause.

As regards prognosis, there is no marked difference between the two classes of cases—"the fact remains that in 200 cases taken haphazard, carefully investigated and followed up, the prognosis as to the future moral and mental condition of the child does not appear to be any better when the first fit is associated with a reflex cause which can be removed than when it is of idiopathic origin."

A. DINGWALL FORDYCE.

**HYSTERIA FROM THE POINT OF VIEW OF DISSOCIATED  
(477) PERSONALITY.** M. PRINCE (of Boston), *Journal of Abnormal Psychology*, Oct. 1906.

THE author's object is to study hysteria from the view-point of dissociated personality, and conversely dissociated personality from the view-point of hysteria. Hysteria is used to denote the type of case which shows on physical examination anæsthesia, paralysis, limitation of the visual field, convulsions, etc., and on mental examination amnesia, irritability, emotionalism, instability, suggestability, etc. In cases of dissociated personality the hysterical symptom-complex may make up one or more of the phases of the multiple individual. In order to show more clearly the contrast in clinical symptoms, 20 cases of dissociated personality are tabulated according to the number, designation, and origin of personalities; general health; stigmata; amnesia; memory, and general character. Upon the analysis of these cases the author concludes that certain complexes with or without amnesia, which are commonly known as hysteria, may be regarded as cases of dissociated personality. They may be considered as phases of multiple personality if taken in connection with the normal condition. Hysteria is a manifestation of disintegration, and the neurasthenic state—one of the stigmata of hysteria—is pathologically a type of dissociation of personality. Conversely, disintegrated personality is no bizarre phenomenon, but in its mild forms an almost every-day clinical affair, though ordinarily, in consequence of the absence of amnesia, it passes unrecognised.

C. H. HOLMES.

**A CASE OF OSTEITIS DEFORMANS WITH HUNTINGDON'S  
(478) CHOREA.** MACKAY, *Lancet*, Sept. 22, 1906.

THE writer merely puts on record a case which showed these two conditions combined, similar cases having been noted elsewhere, to which he gives references.

The patient's memory and intellectual faculties were much impaired and the speech slurred. The muscular power was normal, but there was marked inco-ordination of the limbs, lower jaw, and tongue, and the muscles showed choreiform spasms as well as fibrillary twitchings. Many of the reflexes were absent. The bones of the arms, the ribs, and the femora and tibiæ were much thickened. There was coxa vara, and one leg was much longer than the other and considerably bent. The state of the patient in both respects was getting steadily worse at the time of writing.

JOHN D. COMRIE.

**OPHTHALMOPLÉGIA INTERNA UNILATERALE, WITH  
(479) SPECIAL REFERENCE TO ITS ETIOLOGY AND CLINICAL  
SIGNIFICANCE. EDWIN BRAMWELL and ARTHUR H. H.  
SINCLAIR, *Scot. Med. and Surg. Journ.*, December 1906.**

THE authors in this paper record six cases of unilateral internal ophthalmoplegia.

CASE I. A watchmaker, aged 34, unilateral internal ophthalmoplegia in right eye of three or four months' duration. Syphilis six years previously. Argyll-Robertson pupil on left side. No other indications of nervous disease. Since the symptoms appeared in the eye which he employed almost exclusively when at his work, the case might be cited as an instance supporting the "Ersatz theorie" of Edinger. It is quite possible, however, that the affection of the right eye was merely a coincidence, and that it was the inconvenience produced thereby in connection with his very special work which had compelled the patient to seek medical advice.

CASE II. Left internal ophthalmoplegia in a case of cerebral syphilis.

CASE III. An unmarried lady, aged 38, with left internal ophthalmoplegia. Argyll-Robertson pupil on right side. Absence of both tendo-Achillis jerks. No history suggestive of syphilis or further indications of an organic nervous affection.

CASE IV. Unilateral internal ophthalmoplegia of several years' duration in a male, aged 40, an applicant for life insurance, who presented no further indications of disease.

CASE V. A bank clerk, aged 30, with progressive tabes dorsalis. History of a chancre six years previously. First indication of present illness was dimness of vision and dilatation of left pupil eighteen months ago. Accommodation not examined at that time, but six months later found to be defective in left eye. Pupils regained normal size, and defect of accommodation recovered from under anti-syphilitic treatment.

CASE VI. Left internal ophthalmoplegia of some weeks' duration in a married woman. No evident cause, but a history of miscarriages following marriage was suggestive of syphilis.

Unilateral internal ophthalmoplegia may be due to local trauma when it is almost always associated with dislocation of the lens or other evidence of injury to the eye. Again, it may follow a general injury, as in a case reported by Donath. In some cases of third nerve palsy, dilatation of the pupil and defective accommodation may persist after the external ocular palsy has been recovered from. "Cold" is probably an occasional cause, as in one of Uhthoff's cases. The very great majority of cases of ophthalmo-

plegia interna unilateral *persistens* are, however, associated with syphilis. Alexander has reported twenty-eight cases, in only four of which syphilis could be excluded. Among Uhthoff's cases there are several in which no cause could be detected. All of these cases occurred in young adults. In all there was a well-marked neuropathic factor, while in all, with one exception, the disturbance of vision and mydriasis seem to have disappeared quickly, a point which may help to distinguish this group from the syphilitic cases, in the majority of which the symptom appears to be permanent.

The authors point out that the sign may be of importance in relation to life insurance, since a considerable proportion of these cases—it is impossible as yet to hazard an opinion as to the percentage—ultimately develop parasyphilitic affections of the nervous system. Further, they point out that this is an objective sign of organic disease of the nervous system which it is possible to produce artificially, a circumstance which it may be of use to bear in mind in connection with medico-legal cases.

Schulz concludes, from experimental evidence, that internal ophthalmoplegia, which is a consequence of an affection of the ciliary ganglion or short ciliary nerves, may be distinguished from that which is caused by a lesion on the cerebral side of the ganglion by the fact that the pupil in the latter case contracts under physostygmmin, while in the former case it fails to do so. In several of the author's cases the pupil contracted well under eserine, an observation which suggests, granting that Schulz's conclusion is correct, that the lesion in these cases was situated on the proximal side of the ganglion. EDWIN BRAMWELL.

**INTERMITTENT CLAUDICATION OF THE CORD.** (*La Claudication Intermittente de la Moelle.*) PAUL SOLLIER, *La Presse Médicale*, Oct. 24, 1906, p. 677.

THE recent work of Déjérine (*Revue Neurolog.*, April 30, 1906) on this subject is resumed. It will be remembered that Déjérine has called attention to the features that distinguish it from the intermittent claudication described by Charcot, which is due to a peripheral arteritis. These features briefly are: In the cord affection, persistence of the arterial pulsation in the lower extremities, absence of cyanosis, coldness or any vaso-motor trouble, increase of the patellar and Achilles reflexes with occurrence of Babinski's sign, and, most constant of all, presence of bladder symptoms, usually of the nature of precipitancy; in the muscle affection, abolition of arterial pulsation in the lower limbs, occurrence of vaso-motor symptoms, absence of any symptoms indicating a cord

lesion. In both instances there are no abnormal symptoms or signs except following exertion.

Sollier has met with a typical case, and records it at length. The patient was a man of fifty-four, who had had symptoms for over seven years. The only special features were the occurrence of a relapsing diplopia, and the fact that the upper extremities were affected equally with the lower. Great amelioration was obtained by mercurial treatment, as is usual in these cases. It is because the prognosis is graver, and the effect of treatment more marked, that it is especially important to recognise this group as distinct from that described by Charcot. ERNEST JONES.

**THE CEREBRAL ELEMENT IN THE REFLEXES, AND ITS**  
(481) **RELATION TO THE SPINAL ELEMENT.** G. L. WALTON  
and W. E. PAUL, *Journ. of Nerv. and Ment. Dis.*, Nov. 1906,  
p. 681.

THE authors draw attention to the unsatisfactory nature of the theory that the deep reflexes depend on the integrity of a spinal arc, inhibited in health by a cortical arc, and allowed to become over-active when the pyramidal tract is injured.

The paper is based on sixteen cases of lesion of the brain or cord in which observations were made on the reflexes, and which led the writers to the following conclusions:—

Both the brain and the cord must play a part in the production of the deep reflexes. The higher arcs tend to produce a more subdued, the lower arcs a more violent, reflex. In health the resultant is a comparatively moderate reflex, varying in individuals as the cerebral or spinal type predominates. In organic disease, partial withdrawal of cerebral influence causes exaggeration of the reflex. The spinal arc cannot sustain a reflex alone, since complete withdrawal of cerebral influence causes abolition of all reflexes.

Further, the control of the superficial reflexes differs from that of the deep. They disappear in cerebral disease, and are therefore controlled by the cerebrum, and they are not increased in pyramidal disease, and are therefore not represented in the cord.

The Babinski response is controlled more in the manner of the deep than the superficial reflexes.

Of the cases quoted, six were hemiplegias of rapid onset, and were observed at periods varying from one hour to "some days" after they occurred.

In all, the knee-jerk on the paralysed side was diminished or absent; in two the ankle-jerk of that side was noted as diminished, in two the abdominal reflex was diminished, and in two Babinski's sign was obtained.

Three cases are quoted in which there was increased deep reflex on the paralysed side, and in one of them diminished abdominal reflex was noted.

Two cases of transverse lesion of the cord showed loss of both deep and superficial reflexes below the lesion, without a true Babinski.

Two cases of hemiplegia, with well localised lesion, showed increase of deep reflex in the parts less affected, and diminution in those most damaged on the paralysed side.

These observations go to confirm the cerebral element in the deep reflexes. This element accounts for the abolition of reflex in cerebral disorder, such as epilepsy or chorea, far better than any theory of "irritative inhibition."

Temporary abolition of deep reflex is the rule in apoplexy; when it is retained, cerebral influence has not been completely cut off; the writers suggest that the ultimate increase is due to the uncrossed pyramidal fibres, and is of spinal type.

The work of previous writers on the subject is passed in review, with special reference to Pandi.

D. W. CARMALT JONES.

**A NEW METHOD OF INVESTIGATING THE KNEE- AND (482) ANKLE-JERKS.** (Ueber ein neues Verfahren zur Untersuchung des Patellar- und Achillessehnenreflexes.) FEIX, *Wien. klin. Woch.*, Oct. 11, 1906, p. 1223.

IN examination of the tendon jerks relaxation of the corresponding muscles is indispensable. An objection to the usual methods employed, such as Jendrassik's, is that patients, especially those that have been subjected to frequent examinations, involuntarily make their muscles tense. Feix's method is to make the patient adopt the lateral decubitus with the hip- and knee-joints slightly flexed, and the eyes closed. Complete relaxation of the quadriceps, extensor, and calf muscles is thus obtained, and the patient cannot now make tense his muscles without considerable inconvenience to himself. A further advantage of this method is that both the knee- and ankle-jerks, and even the gluteal reflex, can be examined while the patient remains in the same position. J. D. ROLLESTON.

**A SIMPLE DEVICE FOR OBTAINING THE KNEE-JERK.** (Ein (483) einfacher Kunstgriff zur Erzeugung des Knie-Phänomens.) KRÖNIG, *Berlin. klin. Woch.*, Oct. 29, 1906, p. 1421.

IN cases where the knee-jerks are difficult to obtain, some patients are not sufficiently intelligent to carry out Jendrassik's method.

Krönig has therefore devised the following method, which, like Jendrassik's, is based on the principle of diverting the patient's attention. Directly the doctor says "Now!" the patient is to take a forced inspiration, and at the same time to look up at the ceiling. As the percussion of the tendon must exactly correspond with the inspiration, the tendon must not be tapped until after the word "Now!" has been pronounced, otherwise the patient will delay his inspiration and the jerk will be impeded.

Krönig has carried out this method in some hundred cases and found it most effective.

J. D. ROLLESTON.

**SUBCORTICAL APHASIA (PURE APHASIA).** (*Revision de la (484) question de l'aphasie: que faut-il penser des aphasies sous-corticales (aphasies pures)?*) PIERRE MARIE, *La Semaine Médicale*, Oct. 17, 1906, p. 493.

In this paper Professor Marie continues his revision of the question of aphasia, and deals with the subcortical or pure aphasias. By this phrase is signified the result of a lesion which does not involve the cortex, but which interrupts fibres coming from motor, auditory, or visual centres, and so isolates them from their physiological connections. Pure word-deafness, pure word-blindness, pure motor aphasia, may be specified in this group.

1. Pure word-deafness, according to Déjérine, is the result of a bilateral lesion of the temporal lobes, but from both the clinical and the pathological point of view Marie denies the reality of its existence, for the following reasons:—

He has never seen a clinical case which approaches the condition, and the reported cases do not satisfy him. He affirms the impossibility of its occurrence in a pure form, *i.e.* without any concomitant diminution of the intellectual faculties, and with the other allied speech functions intact. Pathologically, no one of the published cases bears out the reality of the pure subcortical lesion. If any of these cases showed slight deafness, the assumption was made that this was due to a lesion of a cortical auditory centre in the first temporal convolution, but Marie asserts that such deafness is always the result of disease somewhere in the peripheral auditory apparatus.

He suggests that in the so-called auditory word centre there are stored up images not of words, but of syllables; that this centre is therefore an intellectual one, since a complicated intellectual process would be required to reconstitute these syllables into words; and he holds that this explains the frequent defect in all forms of language, however slightly Wernicke's zone is affected. If a patient with word-deafness does not understand what is said to him, it is not because he is deaf to words as words, but because of



the defect of comprehension produced by the lesion of an intellectual (not a sensory) centre.

2. The case is different with pure word-blindness. There can be no clinical doubt of the existence of cases of pure alexia. Déjérine considers the lesion one which destroys the association fibres uniting the common visual centre (bilateral) to the left angular gyrus, the centre for the visual images of words.

But the arguments against the existence of this centre (sensory) are, for Marie, as cogent as those against the existence of a sensory (as opposed to intellectual) auditory word centre. In addition, the fact that the faculties of reading and writing are of quite recent development in the history of mankind militates against the accepted view.

Pathologically, the essential point to remember is that the lesion which gives rise to "pure" alexia is in an area supplied by the posterior cerebral artery, not by the Sylvian. Right homonymous hemianopia is therefore of very frequent occurrence in cases of pure alexia. It is not sufficient for the lesion to involve fibres from the visual zone: the white matter below the zone of language must also be implicated. An affection of the lingual and fusiform lobes in the inferior aspect of the brain will produce "pure" alexia. On the amount of involvement of the fibres coming from Wernicke's zone depends the degree of alexia. Marie is almost inclined to say that aphasia, clinically, depends rather on the distribution of cerebral arteries than on the topography of cerebral convolutions. Pure alexia is really an "extrinsic aphasia": it is a sort of visual agnosia.

3. Subcortical motor aphasia—or pure motor aphasia—is, according to Déjérine, secondary to a lesion which destroys the fibres uniting Broca's convolution to bulbar nuclei. The clinical picture is familiar, and Marie accepts it as of frequent occurrence; its pathological interpretation, however, is another matter.

According to Marie, these are really cases of anarthria, and the anarthric is *not* an aphasic. Broca's aphasia is aphasia plus anarthria; and although Déjérine maintains that dysarthria or anarthria is the result of disease of the peripheral speech mechanism—lips, tongue, palate, etc.—Marie affirms that a patient may be anarthric without any paralysis of the muscular organs of phonation. His anarthria is a functional disturbance, attributable, as the author has already endeavoured to prove, to a lesion in the zone of the lenticular nucleus. Further pathological evidence is offered to show that Broca's aphasia is really anarthria plus aphasia, and a case reported by Déjérine as one of pure motor aphasia is re-analysed, and demonstrated to be one of aphasia from a lesion in Wernicke's zone plus anarthria from a lesion in the lenticular nucleus.

S. A. K. WILSON.

**PSYCHIATRY.****THE EARLY DIAGNOSIS AND TREATMENT OF PROGRESSIVE**

(485) **PARALYSIS OF THE INSANE.** (Frühdiagnose und Behandlung der progressiven Paralyse.) KARL HEILBRONNER, *Deutsche med. Woch.*, Oct. 4, 1906, S. 1609.

MOST of the cases sent to institutions with the diagnosis of early G.P.I. are fairly advanced, as the early symptoms are not sufficiently studied. One is not justified in making the diagnosis until both aspects of the disease, the dementia and the paralysis, shew signs of their presence. At the same time these signs may be so very faint as to require special investigation directed towards them before they can be observed. The dementia is much less obvious in the case of women and children. It often begins in the moral sphere, so that a previous knowledge of the patient is necessary in order to detect it. Unmannerliness, disorderly dress, the sudden appearance of a love for a public-house bar, are all suspicious symptoms; at the same time fleeting spasms of remorse or of rage are often noticed. Amongst the intellectual symptoms, deficiency in attention, forgetfulness—usually of the continuous amnesia type—ignorance of simple facts, are prominent. Hypochondriac-depressive moods, alternating with euphoria, are often noticed. On the physical side, indication of pyramidal disease, such as Oppenheim's and Babinski's signs, of posterior column implication, and disturbances of micturition, are to be looked for as shewing cord affection; and pupil changes, ataxy, tremor, and changes in facial appearance and in speech, as shewing cerebral affection. The above are described in an elementary way, and no new points of view demonstrated by the author. The indications for sending the patient to an asylum are mentioned. In addition to dangerous symptoms, other indications are great excitability, expansive delirium, a hypochondriac condition.

ERNEST JONES.

**SECOND NOTE ON FALSE REMINISCENCE.** (Deuxième note (486) sur la fausse réminiscence.) FÉRÉ (of Bicêtre), *Journ. de Neurol.*, mars 20, 1906, p. 101.

A CASUISTIC contribution to the study of the phenomenon of false reminiscence, *i.e.* the false impression of having already seen, heard, or experienced things. The first patient was a neurasthenic woman with paræsthesia of the extremities, transitory paralyses at night, various visual disorders, nocturnal episodes of awaking

with a feeling of great anguish. Transitory dimness of vision was frequently followed by the illusion of having previously seen an object, *e.g.*, she said to the fishmonger, who had not called before, "You showed me yesterday that lobster."

In the second case, the phenomenon occurred in a man convalescent from grippe; it was associated with a state of fatigue, and disappeared with the latter. C. MACFIE CAMPBELL.

**THE POLYNEURITIC PSYCHOSIS AND BERI-BERI** (*La psychose (487) polynévritique et le béribéri.*) NINA RODRIGUES (of Brazil), *Ann. Méd.-Psych.*, March-April 1906.

MANSON has denied the presence of the syndrome of Korsakow in polyneuritis, due to beri-beri, and emphasises the absence of memory defect as a differential point between a polyneuritis of this origin and a malarial polyneuritis. The author contradicts this view absolutely, basing his views on observations in Brazil; he reports fourteen cases, and gives the following conclusions. Korsakow's polyneuritic psychosis occurs in polyneuritis caused by beri-beri. Taking the three forms of Korsakow's psychosis grouped by Ballet as amnesic, delirious, confusional, the amnesic form is the most common. Sometimes the amnesia is much more far-reaching than the amnesia of Korsakow's psychosis. Absence of amnesia is not of differential value. C. MACFIE CAMPBELL.

**MILD FORMS OF DEMENTIA PRÆCOX.** (*Les formes frustes de (488) la démence précoce.*) J. CROCQ (of Brussels), *Journ. de Neur.*, April 5, 1906.

THE author starts from the heboidophrenia of Kahlbaum, which consists in a slight reduction in the various mental faculties at puberty; the adult does not fulfil the promises of his youth, his interests and abilities show slight deterioration, but there is neither delusional formation nor excitement. It is a mild form of Hecker's hebephrenia. Crocq notes the deterioration in interests, character, occupation and behaviour of this group. He next reports the case of a patient with bad heredity, always slightly peculiar, who, at the age of twenty-nine, showed increased want of balance and change of character; he became irritable, neglected his business, was odd, occasionally mute and refused to eat, talked of suicide. This ill-balanced behaviour was in part precipitated by the failure of his matrimonial plans.

When committed, the main feature was his ready acceptance of hospital *régime*, with no desire for any wider sphere of interests ; he was not depressed, amused himself with the others, and on discharge he continued to lead an equally limited existence.

Such a case is considered by Crocq to be intermediate between heboidophrenia and a third form, with short delusional attacks, from which the patient recovers with comparatively slight defect ; after a series of such attacks the patient gradually descends the mental scale. As examples of this form he reports the observations of two patients with bad heredity and constitutionally defective. The first had recurrent maniacal attacks, and was observed during an attack at the age of forty ; he had numerous absurd delusions. After two and a half months he left the hospital convalescent, but still showed a paranoic attitude. Unfortunately the report lacks definition, especially with regard to the differentiation of the attacks from those of manic-depressive insanity. The second case had periodic attacks of "maniacal incoherence" ; on admission, at the age of forty-six, he was excited, incoherent, gesturing ; made a series of absurd statements. After five months he left convalescent, and continued to lead an ill-balanced life.

The author does not give any valid reasons for bringing such cases into the group of dementia præcox, he does not discuss their relation to the periodic psychoses, nor consider the question of constitutional mania.

C. MACFIE CAMPBELL.

**MANIC-DEPRESSIVE INSANITY.** (*De la folie maniaque-dépressive* (489) *sive.*) G. DENY (of Paris), *Arch. de Neur.*, July 1906.

IN this lecture Deny gives a brief but clear historical account of the steps which have led to the formation of the clinical group of manic-depressive insanity. The earliest writers noted the alternation in certain patients of maniacal and depressed attacks, but mania and melancholia were regarded as two distinct entities before the works of the French school showed their intimate relation ; this first period closes with the publication in 1851 of Falret's first communication on the subject.

The second period is that of the French school ; Falret and Baillarger called attention to cases where the attacks consisted of more than one phase, a maniacal phase passing into a depressed phase, and a clear interval either separating the two or following the depressed phase.

This new clinical entity, which was separated from the classical mania and melancholia, was called by Falret circular insanity, while Baillarger named it "folie à double forme."

Kraepelin is the representative of the third or German period. He called attention to the fact that so-called simple mania, if followed for a long period, was found to recur; the simple melancholia—from which he separated certain depressions in advanced life and other depressions forming part of a deteriorating psychosis—also recurred or alternated with periods of excitement.

He therefore brought together simple mania, simple melancholia, circular insanity, recurrent mania, and melancholia into the one large group of manic-depressive insanity. Deny justifies the term manic-depressive not only for recurrent cases with the two phases, but for all cases; the series of attacks may show predominance of one phase, but the other phase may occur later. Even in the individual attack, symptoms of the opposite phase appear either faintly indicated, or well enough marked to make it useful to group the case as belonging to a mixed type. The author accepts fully Kraepelin's views, and defines the psychosis as a constitutional disorder, essentially hereditary, characterised by the repetition, alternation, juxtaposition, or coexistence of states of excitement and of depression; it is a clinical entity belonging to the large group of constitutional psychoses.

C. MACFIE CAMPBELL.

**THE FORENSIC IMPORT OF SEXUAL PERVERSITY.** (*Die (490) forensische Bedeutung der sexuellen Perversität.*) J. SALGÓ (Budapest), *Sammlung zwangloser Abhandlungen aus dem Gebiete der Nerven- und Geisteskrankheiten*, Bd. vii., H. 4, p. 43. Halle a. S.: Carl Marhold. 1907. Price M. 1.20.

THE present paper is confined to an examination of homo-sexuality and an inquiry into the various legal restrictions placed upon unnatural offences, and the practical results of the operation of these laws. The author reviews the various laws of different countries, and points out their disparity in this respect; some countries, notably France and Japan, taking no particular legal cognisance of these practices. With the exception of Hungary, however, all countries which do legislate in this matter, punish only male homo-sexuality and sodomy, and even in Hungary sapphism is in practice disregarded. In the author's opinion, the law stipulates either too much or too little. On the one hand, if the punishment is intended to enforce sexual congress as entirely a procreative act, then many other practices should come under the ban of the law, which as it stands is thus too limited; and on the other hand, if the general execration of sexual inversion is rooted in an aversion from their unnatural or abnormal character,

the law, in so far as it takes notice of secret vices which do not violate public decency, oversteps its bounds and infringes private interests. The author therefore considers that it would be better if the various articles dealing with homo-sexuality were expunged from the law and its punishment left—as are already many other immoral or vicious acts—to social condemnation.

From the point of view of psychiatry Dr Salgó finds nothing in these phenomena to justify such a term as “psychopathia sexualis.” Homo-sexuality may be a symptom, a *Teilerscheinung*, of one or other morbid process, but in itself gives no indication of any type of disorder.

R. CUNYNGHAM BROWN.

**THE REGULATIONS AS TO THE DISCHARGE OF PATIENTS (491) FROM ASYLUMS IN PRUSSIA.** (Die in Preussen gültigen Bestimmungen über die Entlassung aus den Anstalten für Geisteskranken.) Professor C. MOELI, of Berlin. Pp. 44. Halle a. S. : Carl Marhold. 1906.

PROFESSOR MOELI furnishes in this short paper an exceedingly interesting commentary on the working of the provisions made in the Bürgerliches Gesetzbuch with regard to the discharge of patients from asylums. As the German lunacy laws differ fundamentally from our own, his criticisms of Prussian law are of only relative interest. Nevertheless the same problems which confront English medical superintendents are being widely discussed in Germany, such as, for example, to instance only one or two of these, the question of the discharge of recovered patients whose subsequent relapse seems inevitable; the provision of some form of supervision and after-care of patients having no legal guardians; and—a difficulty which is naturally much more sorely felt in Germany than with us—the ill-effects of maintaining criminal lunatics in ordinary public asylums. These are among the points ably and temperately discussed by Professor Moeli. His conclusions are, of course, inapplicable here, with perhaps one exception, namely, the formation of an after-care association similar to the German equivalent for our Discharged Prisoners' Aid Society. Doubtless if the sphere of operation of the English After-Care Association could be so widened as to exercise for a prolonged period some supervision over and personal interest in all rate-aided patients discharged from asylums, whether requiring pecuniary assistance or not, their discharge would be facilitated, and, quite possibly, the chances of their relapse diminished.

R. CUNYNGHAM BROWN.

**TREATMENT.**

**POTASSIUM BROMIDE IN EPILEPSY.** (*Emploi du bromure de (492) potassium dans l'épilepsie.*) JULES and ROGER VOISIN, *Presse Médicale*, Aug. 25, 1906, p. 541.

THE mechanism of the action of bromide in epilepsy is that it is supposed to take the place of the molecules of sodium chloride, which are combined with albuminoids. Various adjuncts have been proposed in its administration. The authors recommend pilocarpin, and give the following prescription:—

Potassium bromide . . .	70 grammes.
Nitrate of pilocarpin . . .	·035 "
Syrup of orange . . .	400 "
Water . . .	600 "

Pilocarpin is useful, because of its diuretic and sudorific action. "Dechloridation," or the reduction of the chlorides, is not always advisable, however much its worth has been vaunted. The regime which the authors have found serviceable may thus be indicated:—

- 10 days of ordinary diet, with 4 grammes of KBr daily;  
followed by
- 10 days of ordinary diet, with 10 grammes of KBr daily;  
followed by
- 10 days on a salt-free diet, and no bromide.

This sudden suspension of the bromide is harmless, for the latter has soaked the tissues of the organism, and does not disappear at once from the body. On the other hand, it is a mistake to suspend the bromide if the patient has previously been on a salt-free diet.

S. A K WILSON.

**THE PRESENT STATUS OF BRAIN SURGERY.** ALLAN STARR, (493) *Journ. of Amer. Med. Assoc.*, Sept. 22, 1906.

STARR regards the present time as a suitable one for coming to final conclusions in regard to the propriety of operations on the brain, since it is twenty years since the first operations were performed. He thinks that it is now more possible to take a broader and calmer view of the entire subject; to realise the limitations of such operations; to appreciate their true value, and to determine with exactness the class of cases in which brain surgery promises success. He derives his conclusions from an extensive experience in this field, and does not lay too great stress upon statistics, because

he realises that very many cases, both of a favourable and unfavourable nature, have not been published.

With regard to the surgical treatment of epilepsy, he thinks that the cases of epilepsy which are open to surgical treatment are those in which it can be determined that there is a localised focus of disease in the brain which acts as a starting-point for irritation, and thus causes the epileptic attack. Such local foci of irritation may be produced by an injury to the head not attended by external evidence of injury. Internal causes, such as a small local inflammation of the meninges, a plaque of sclerosis in the brain, the origin of which may be wholly unknown, are capable of producing epilepsy. He points to experience in the treatment of abscess and tumour as showing that the necessary cutting of the brain for the relief of these diseases is liable to result in the production of gliomatous scar tissue, which subsequently becomes a focus of irritation, leading to epileptic attacks. He points out that the essential difference between epilepsy produced by these local lesions and idiopathic epilepsy consists in the fact that in the cases open to operation the epileptic fit is usually preceded by a single kind of aura, either a sensation of numbness in one extremity, or a hallucination of sight, sound, smell, or taste; or by a sensation of muscular twitching, which is immediately followed by a localised spasm, confined at first to one extremity, or to one side of the face, and extending, if it does extend, in a definite manner to the other parts of the body. The fit is rarely attended by a loss of consciousness. He says that such a localised epilepsy, generally described as Jacksonian epilepsy, is always due to a definite focus of disease in the brain; furthermore, that this disease is cortical, and hence accessible to the surgeon; and that, by following the well-known guides to the localisation of such diseases, it can be excised, either from the meninges or from the cortex, when these parts are laid bare. He distinctly states that these are the only cases of epilepsy which are open to cerebral operation. He points out that epilepsy is a widespread disease among the community, one person in five hundred being affected, and in his experience not more than 2 per cent. of the cases of epilepsy are in any way open to operation, the other 98 per cent. presenting none of the characteristic symptoms of localised disease in the brain. He thinks it is useless to trephine idiopathic epilepsy, even though it may apparently have been caused by a fall or a blow on the head, unless the attacks present the characteristics here named. He thinks the prognosis in the cases of focal epilepsy open to operation is not so favourable as was at first anticipated; even when a focus of disease can be excised from the brain, or a plaque of adherent meninges loosened or taken away. Some cicatrix is inevitably left, and this too often remains as an adequate focus for



the recurrence of the epilepsy. In only about 20 per cent. of the patients operated upon has permanent cure been the terminal result. In many cases relief for a certain time has followed the operation, but after one, two, or three years these patients have returned with a renewal of the symptoms, and second and third operations have not been followed by permanent cure. Such unfavourable results might possibly lead to the conclusion that in the majority of these cases of focal epilepsy the operation should be refused. But, as Starr points out, it is often found that the focal epilepsy is the first symptom of the growth of a tumour, or the development of a cyst in the cortex of the brain, and a careful study of the histories of the cases of brain tumour reveals the surprising fact that in many of them focal epilepsy was the earliest sign of the development of the disease. He says that it is therefore imperative to operate in these cases of focal epilepsy as soon as the diagnosis is made: (1) because of the possible relief of epilepsy; and (2) for the possible discovery of a more serious and fatal disease, which is just commencing.

He reports operations performed for the relief of epilepsy of a focal nature in about sixty cases. In a few of these relief by operation was found to be impossible. In about one-fifth a permanent cure was obtained. In the remainder the epilepsy recurred, and was in no way affected by the operation. He concludes as a result of his experience, that the operation of trephining in epilepsy is of very limited application, and is only to be recommended in a few selected cases, which present the necessary guides to both physician and surgeon.

Turning to the surgical treatment of abscess of the brain of traumatic origin, he thinks that they should be operated upon early, when located in parts of the brain which produce definite cerebral symptoms. In cases of fracture of the skull, or concussion, followed within two or three weeks by the development of symptoms suggestive of abscess, Starr says that it is imperative to trephine, even though the indications are purely those of a surgical kind, there being no localised cerebral symptoms; for there are many districts of the brain, disease of which does not produce known effects.

Patients with abscess of the brain, developed subsequently to chronic otitis media, should be operated upon as early as it is possible to make a diagnosis. He thinks the diagnosis of abscess of the brain is comparatively simple in cases in which the usual causes have preceded the development of surgical symptoms. Headache, vertigo, vomiting, slow pulse, marked change in the mental state, sensations of dulness and hebetude, slowness of thought, irritability of temper, defective memory and depression, tenderness of the head to percussion, irregular pupils, and optic neuritis, constitute sufficient evidence of a cerebral complication.

When meningitis occurs, as a rule lumbar puncture will reveal an increased number of leucocytes in the cerebro-spinal fluid, together with the existence of micro-organisms. He therefore regards lumbar puncture as an important means of differential diagnosis between meningitis and cerebral abscess. He regards the examination of the blood also as a means of diagnosis, for a sudden and great increase in the leucocytes is an indication of a cerebral complication, and the leucocyte count in meningitis is usually higher than that in abscess. Starr holds that as soon as the diagnosis of cerebral abscess is made under any circumstances, it is imperative to operate; and while the percentage of recoveries, from the nature of the case, is at present only about 60 per cent. in the statistics that he has collected, including about 500 cases, yet he holds that there is every reason to believe that in cases which are diagnosed early, and operation undertaken as soon as the diagnosis is made, the percentage of recoveries will be much greater.

Turning to the surgical treatment of tumours of the brain, he thinks that in cases in which the diagnosis of brain tumour is made early from the general symptoms, together with the special localising signs, operative interference is warranted; but that in the far larger number of cases in which localising signs are absent, operation promises nothing. He refers to the statistics published by Knapp, which the author of this article has already reviewed in the last number of this journal, and finds that they are quite in accordance with his own experience. He thinks that, even though the number of lives saved is a small one, it is our duty to study every case with our minds fixed on the question of possible surgical relief. He thinks that palliative operations for the relief of symptoms are justifiable, although he quotes two cases in which they did not succeed.

Referring to the surgical treatment of cerebral hæmorrhage, he thinks that it requires a large amount of courage to suggest an operation on the head of a patient who is comatose from an apoplexy. He refers to the fact that Harvey Cushing has applied successfully in hospital cases the test of the condition of the pulse tension, in order to determine the need of surgical intervention to prevent death. When the blood pressure rises steadily to 250 mm., measured on the Riva Rocci apparatus in a case of apoplexy, and coincidentally with this rise a very slow pulse, falling to 50 a minute, we may say that the case will be fatal. He thinks that in such a case we are justified in resorting to surgery. The object in view is to relieve the intracranial pressure. This is done by turning down a large bony flap, or making a large fenestrum in the skull, irrespective of any attempt to find or remove the clot. The best place to open is over the motor area on the side opposite

to the paralysis, as in some cases the clot may be found there. The dura should be exposed and divided. If the clot is on the surface, it will then be seen. If the clot is within the brain, the brain will bulge, and division in the depth of a fissure may succeed in reaching and evacuating the clot. Even if the clot is not found and removed, the relief of the intracranial pressure stops the alarming symptoms, and recovery is much hastened, as Cushing's cases have proved. The pulse tension falls as soon as the skull is opened. The pulse becomes more rapid, Cheynes-Stokes respiration ceases, and consciousness soon returns. The operation thus offers life in a formerly hopeless condition. He refers to Cushing's operations in cases of hæmorrhage in the new born, and thinks that any measure for their relief is justifiable. He thinks that Cushing's success in these cases warrants us in urging on all obstetricians the duty of considering this operation in asphyxiated infants. It is they who see these cases, and if they can be persuaded that delay in them is dangerous, and the prospect of relief is good, the percentage of idiocy and hemiplegic epilepsy will be reduced.

He refers to the cases of cerebral hæmorrhage, in which hemiplegia, aphasia or hemianopsia, develop slowly after an injury, and do not come to their height for three or four days, as another class of case open to trephining. A lumbar puncture will reveal bloody cerebro-spinal fluid. The symptoms may increase and threaten life, as shown by the slow pulse, Cheynes-Stokes respiration, and increasing blood pressure; or they may come to a standstill, leaving the patient permanently incapacitated. In either case surgical treatment is indicated.

He refers to a successful case, operated on in 1889, and many other cases, which have been equally successful, since then.

Passing on to the surgical treatment of imbecility, due to microcephalus, he refers to the operation of craniectomy, for allowing an increased expansion of the brain. In spite of many operations, reported in many lands, he says that the subsequent history of these cases has demonstrated conclusively that no marked mental improvement ever follows this operation. It is evident that the microcephalus is not the cause of the imbecility, but the imperfect and rudimentary development of the brain, which does not grow in proportion to the rest of the body, prevents the normal expansion of the skull over a normally growing brain, and leads to an early closure of the fontanelles. He says that the slight improvement which in some cases has appeared to follow the operation, in no way differs from a corresponding improvement in patients equally affected, and equally trained, who have not been operated upon. For this reason he no longer recommends any operation on the head in cases of idiocy, im-

becility, hemiplegia, and epilepsy, dating from childhood, and states that these operations have no effect whatever on the painful condition so often associated with these conditions, namely, athetosis.

DONALD ARMOUR.

**THE SURGICAL TREATMENT OF TRIGEMINAL NEURALGIA.**

(494) Being a Study of the Cases of Recurrence after Operative Treatment, with Suggestions as to the Best Methods of Obviating Post-Operative Recurrence. MOSCHCOWITZ, *New York Med. Rec.*, Sept. 29, 1906.

MOSCHCOWITZ shortly reviews the pathogenesis of trigeminal neuralgia. He points out that there is no agreement in the views of the numerous observers as regards the pathology of the disease, and classifies the changes that have been found under two headings:—

1. Those that have been found in the peripheral nerves.
2. Those that have been found in the Gasserian ganglion.

1. The following are the various pathological changes found in the peripheral nerves by various writers:—Dana: an obliterative endarteritis, but no changes in the nerve elements. Putnam: an endarteritis and a general fibrosis. Horsley: a sclerosis, with a shrunken epineurium, probably due to an active congestion and oedema in the early stages. Schweinitz and Rose: an obliterative endarteritis, with the axis cylinders swollen or shrunken, and occasionally absent, the myelin sheaths swollen, the sheaths of Schwann swollen and markedly rich in nuclei. The endoneurium was increased, particularly in the neighbourhood of the blood vessels. The changes resembled those of a chronic neuritis, and as they were more marked at the periphery than at the centre, Rose assumed that the lesion was an ascending one. Krause's examination of nerve trunks, obtained by evulsion, showed only occasionally a thickening of the nerve sheaths, and nothing else.

2. The following are the pathological changes that have been found in the Gasserian ganglion by various observers:—Rose: a thickening of the interstitial connective tissue, and irregularity in the formation of the ganglionic cells. He regards the disease as a chronic inflammatory process. Horsley has found no more degenerative changes in the ganglion than could be accounted for by the age, condition, and habits of the patient. Antonio d'Antona found a sclerosis of the ganglion. Krause found no sclerosis or endarteritis, but only degeneration of the ganglionic cells and nerve fibres. He regards the origin of the disease as being in the Gasserian ganglion. Moschcowitz thinks that the argument that

the disease is a neuritis can probably be negatived, because, with the exception of the symptom of pain, trigeminal neuralgia presents none of the symptoms and physical signs of any other well-recognised neuritis, such as anæsthesia, paralysis, or the electrical reactions. Frazier thinks that, from our present knowledge, there are two or three distinct types of trigeminal neuralgia, one in which the lesion is primarily a neuritis of the peripheral branches, which subsequently may or may not extend to and invade the ganglion; another, in which a primary lesion first appears in the ganglion; and a third, in which the lesion is neither in the ganglion nor its branches, but in the central nervous system. The lesion may be of a degenerative type, an interstitial neuritis, or a neoplasm.

Spiller, from the examination of ten Gasserian ganglia removed by Keen, describes his findings as follows:—

“The lesions in the Gasserian ganglion, in the more advanced cases of *tic douloureux*, consist of large swollen medullary sheaths, swollen axis cylinders, atrophied fibres, empty nerve sheaths, nerve bundles, in which the nerve elements have been destroyed and only connective tissue is left, atrophied ganglion cells, cells faintly stained, and sclerosed blood-vessels, in some cases even without a lumen.”

Moschcowitz points out that all the observers quoted above have regarded the pathological changes as primary, and in direct ætiological relation to the disease. Assuming, then, that many of the changes which they describe have actually occurred, he thinks we cannot accept their findings as primary, because all the pathological changes can be easily accounted for as secondary products. These secondary influences might be classified as follows:—

1. Rough handling of the nerve or ganglion, as a result of the necessary manipulations in the course of the extirpation.
2. Incomplete preservation of the ganglion after removal.
3. The age of the patient.
4. Ascending changes in the nerve or ganglion as the result of previous peripheral operations.

He refers to the researches of Monari, Schwab, and Coenen, as showing the possibility of this. These observers had examined five ganglia, in cases where no previous peripheral operation had been performed, and found the ganglia were entirely normal. Billroth, Hutchison, and Cushing have found no changes in the ganglion or nerve.

Moschcowitz suggests that trigeminal neuralgia may possibly be accounted for by assuming some variety of vasomotor disturbance. He therefore classifies it with such diseases as Raynaud's disease, intermittent claudication of Erb, erythromelalgia, etc.,

although he confesses that this theory cannot be proved by definite pathological or physiological evidence. He regards extirpation of the Gasserian ganglion as a symptomatic cure, and as the treatment of trigeminal neuralgia has resolved itself into the relief of pain, the simplest operation that could be devised would be one which would prevent unnecessary impulses from passing into the brain—in other words, neurotomy. He refers to the first neurotomy, done in 1748 by Schlichting, and others by Louis in 1766, and Viellart in 1768. He refers to the disappointment following the operation for neurectomy, which was a direct development of the first proceeding. He refers to the various operations for attacking the branches of the ganglion at their exit through the foramina at the base of the skull. He says that these operations have been found wanting, because of the frequency of recurrence of the malady in a large percentage of cases, and says that, as in neurotomy and neurectomy, the cause of the relapse has in every instance been due to regeneration of the divided nerve. He thinks that these operations at the base of the skull are merely neurotomies and neurectomies, performed at a higher level, and that the relapses are not to be wondered at. He then refers to Thiersch's operation of evulsion of the nerve (neurexairesis), brought forward in 1889. Recurrences were as frequent after this method as before. The idea of attacking the intracranial portion of the fifth nerve was first suggested by Mears in 1884, who, however, never performed it. After operations by Rose, who used the pterygoid route, and Horsley, who in 1891 divided the intracranial root of the fifth nerve, ganglionic extirpation was placed on a firm footing by Hartley and Krause. Moschcowitz refers to the comparatively high mortality, which he apparently arrives at from cases collected some years ago. He regards the results in a very large percentage of all operative recoveries as excellent. He refers to instances of recurrence reported by Friedreich, Garré, Parmenter, Sapejko, Schwartz, Marchant and Hebert and Perthes. He thinks that on account of these recurrences after complete extirpation of the Gasserian ganglion the operation is not based on a correct principle. He further thinks that he is justified in coming to the following conclusions regarding the Hartley-Krause operation:—

1. The operation is attended by a very high mortality.
2. It undoubtedly gives the greatest number of complete recoveries.
3. Occasionally, though rarely, recurrences have been met with after this operation, even in competent hands.
4. The relapses are due to a regeneration and reunion of the divided nerves.

He concludes his article by advocating the plugging of the foramina after section of the nerve with fine celluloid, such as is

used in photographic films. This suggestion is merely a modification of one made by Abbé, who used rubber tissue as an intervening medium. Moschcowitz also suggests thin flat gold buttons, provided with suitable sized shanks, for insertion in the foramina. He then refers to the intraneural injection of osmic acid, which has been so warmly advocated by Murphy, and points out that in the majority of cases a recurrence follows sooner or later. Moschcowitz sums up the treatment of trigeminal neuralgia in the following conclusions:—

1. Eliminate any possible ætiological factors, such as tumours, carious teeth, antral disease, malaria, syphilis, etc.

2. Determine accurately the nerve branch or branches involved.

3. The operation should be performed as near to the periphery as possible.

4. The operation should be performed early. This is important, because the earlier the case, the more chances there are that a peripheral operation will be of benefit.

5. Whatever the character of the operation may be, the dominant principle must be the prevention of regeneration of the affected nerve.

More specifically, the operation may be classed under two headings, peripheral and central.

6. He believes that if the above principles in the treatment of trigeminal neuralgia are carried out, the operation of extirpation of the Gasserian ganglion will become entirely unnecessary.

DONALD ARMOUR.

#### **SOME POINTS IN THE SURGERY OF THE PERIPHERAL (495) NERVES.** JAMES SHERREN, *Edin. Med. Journ.*, Oct. 1906, p. 297.

A NUMBER of problems are here discussed, and tables of cases referring to each are appended. The first question concerns the methods available to restore continuity to a divided nerve, the ends of which cannot be brought into apposition. These methods fall under five groups:—

1. *Nerve transplantation*, the gap being filled by a portion of nerve derived from another source. Where the source is the patient or another human being, the results are good, perfect recovery being not infrequent; this is the ideal operation. Where the nerve is from an animal, the results are worse, the piece of nerve apparently dying instead of merely degenerating; only one out of sixteen cases was completely successful, and six were partially so.

2. *Flap operations*, a portion of the nerve being turned down or up. Though not without successes, this method is not encouraging.

3. *Provision of an artificial path for regeneration*. Usually strands of catgut are used for the bridge, the nerve ends and catgut being sometimes surrounded by a tube of decalcified bone, or other material, to prevent adhesions. This method gives results equal to those of transplantation of animal nerve.

4. *Utilisation of neighbouring nerves*. Here there are two varieties: (1) Nerve crossing, where the sound nerve is completely divided, and its central end united to the peripheral end of the injured nerve. (2) Nerve anastomosis, where only some of the axis cylinders of the sound nerve are divided and employed. The latter variety practically is the only one suitable for traumatic cases. Nerve anastomosis may be *central*, where the whole of the sound nerve or a slip from it is inserted into the side of the injured nerve; or *distal*, where the whole or part of the peripheral end of the injured nerve is inserted into the sound nerve, or attached to a slip of it. Of twelve cases of anastomosis all but two showed improvement.

5. *Shortening the limit by resection of bone*. This is rarely justifiable.

The second question considered in the article is the treatment of facial paralysis by nerve crossing or nerve anastomosis. The spinal accessory or the hypoglossal may be employed, and in none of the cases collected has some voluntary movement—in many cases very complete—failed to return. The chief difficulty is the tendency for facial movements to be associated with those of the trapezius or tongue, according to the nerve used; but dissociation of the movements has now occurred in a number of cases, the hypoglossal having given the better results in this respect. Transverse incision into the nerve and suture into it of the peripheral end of the facial seems to be the operation of choice. As a rule, improvement does not begin for four or five months, and requires several years for completion, massage and electricity being meanwhile used as may be necessary.

These successes naturally suggested operation on cases of paralysis of central origin, such as infantile paralysis. The results in suitable cases are distinctly encouraging. The best operation seems to be complete division of the affected nerve, and end to end suture to a flap raised from a sound nerve. If the affected nerve, however, be small, it is sufficient to suture it into a slit in the reinforcing nerve.

W. J. STUART.



**FAILURES IN THE TRANSPLANTATION OF TENDONS.**

(496) (*Misserfolge der Sehnenüberpflanzung.*) OSCAR VULPIUS,  
*Berlin. klin. Wochenschr.*, No. 42, 1906.

THE ill-successes attending such operations are chiefly to be attributed to the selection of unsuitable cases. The more limited the paralysis existing, the greater is the prospect of success; for the more antagonistic the action of the muscle employed is, the greater is not only the mechanical disability, but the strain thrown on the central nervous system in accommodating its functions. If the available muscles are paretic, either from previous poliomyelitis or atrophy of disuse, they may not be capable of replacing those actually paralysed, but in course of time become stretched and useless. In such cases implantation of tendons on bone is the preferable operation. One difficulty in ensuring success of transplantations lies in determining beforehand the degree of usefulness or capacity of recovery of the available muscles.

In quadriceps paralysis the results of operations are wonderfully good. In deltoid paralysis tendon transplantation is contraindicated. Arthrodesis gives better results. With regard to the forearm the great risk is of over-correction, and transplantation to correct extensor paralysis of the hand is useless if, as may easily happen, limitation of flexion and of the finer movements of the fingers results.

In the paralysis associated with poliomyelitis transplantation operations must await the termination of the disease and its ultimate effects on the muscles implicated. In the case of progressive muscular dystrophy, however, Vulpius considers that transplantation of tendons is not contraindicated during the course of the disease, if for instance during a pause in its advance one can secure for the patient an improvement in locomotion for the space of a few years.

In spastic affections of children associated with mental weakness, such cases as show paralysis of bladder or rectum, spastic paralysis of the arms or athetosis are best left unoperated on.

In operative procedures care must be taken to avoid in the restitution of one function the complete abolition of another, as for instance by transplanting both peroneal muscles. From the point of view of nerve function it is better to use a whole muscle than to split it. Great care must be taken to ensure the preservation of tone in the transplanted muscle; too great or too slight tension will result in atrophy. Rest must be sufficiently prolonged after operation to procure firm union of the sutured tendons. Massage and exercises are subsequently of prime importance to avoid stiffness and to assist the development of the nervous accommodation.

L. C. PEEL RITCHIE

**REPORT OF THE CONGRESS OF ALIENISTS AND NEUROLOGISTS OF FRENCH-SPEAKING COUNTRIES****Held at Lille, August 1906.***(Continued.)*

THE third report, that on the responsibility of hysterics, was prefaced by its author, Leroy of Ville Evrard, with the quotation of the various definitions of this neurosis. Those of Pitres, Bernheim, Babinski, and Janet were considered, but the reporter neither selected one, nor gave a precise definition of what constitutes hysteria. The physiological theory of Sollier, however, was not cited. As a consequence, the report becomes an attempt to appreciate the responsibility of subjects showing manifestations considered to indicate a morbid condition hitherto so vague as to escape definition. It is only just to add, however, that Leroy attempts to separate from pure hysteria such conditions as mental degeneration and intoxications.

The unsatisfactoriness of this want of precision led the Congress to seek for a report which will enable them to focus the manifold ideas regarding the definition and nature of hysteria, and it may be advisable to defer an analysis of the views on this subject until Claude has presented his report at Geneva.

Given then this ill-defined condition, is any modification of responsibility entailed thereby? This will depend upon two points: (1) the patient, as regards antecedents and physical signs, and (2) the peculiarities of the crime committed. As to the patient, Leroy considers that even slight attacks of paralysis, contracture, or somnambulism, are more serious than syncope with constriction of the throat; and he thinks that a considerable anæsthesia of general, special, or visceral senses indicates grave psychic trouble tending towards double personality.

Quite as important is it, in a medico-legal examination, to appreciate the degree of suggestibility, of aboulia, or of amnesia. A patient showing these symptoms should often be deemed irresponsible for an act which in itself seems to have been dictated by motives that are perverse though not irrational. The responsibility in such cases is qualified as "modified" by most experts, but Leroy considers that such subjects are diseased, and are therefore completely irresponsible on account of their true mental inferiority, which entails atrophy of the altruistic or social feelings along with a restriction in the breadth of apperception. For him confinement, when indicated, should be in an asylum, not in prison, as well in the slight cases as in the grave hysteria which no one disputes.

In the second place, however, the characters of the act often in themselves show the patient to have been at the time irresponsible, even although physical examination may show the stigmata to be slight, while on the other hand an avowed hysteric is not irresponsible for every act he commits. To give such people a bill of indemnity to the detriment of public order is not only inexpedient but inexact.

What conditions, then, indicate irresponsibility? Those acts committed when the subject is under the influence of hallucinations, or of delusional ideas, or is in an ecstatic or agitated state, even when these are merely transitory and more or less abortive; for in the opinion of Leroy, these states are substitutions for convulsive fits, and are the only true hysterical delires. Of course the actions committed during somnambulism are judged in the same way. A theft, a murder, or a flight is here dictated by a fixed idea, of which the patient is the automatic plaything. To the so-called "second state," which is merely a prolonged somnambulism, the same considerations apply. The two personalities may have entirely different mental content, and be different characters. In judging of responsibility, one must be very careful to see that the act has really been committed during the "second state."

This is the explanation of many of the lies of hysterics. Leroy agrees with Pitres in saying that Huchard and Legrand du Saulle were mistaken in attributing to simulation phenomena of this kind simply because they could not explain them. As Janet has clearly shown, the hysterical consciousness is sometimes so vague that dream states are mistaken for reality, and *vice versa*, while the distinction between yesterday and to-morrow is seldom clear. First one image predominates and then another, and hence the untruthfulness; for the hysteric is not an inhibitory creature. His images eventuate in action; impulsion arising in misconception is the foundation of a crime.

But mythomania, as Dupré calls a morbid tendency to lying, is often fully conscious and intentional, and springs chiefly from the desire to create an impression. It is an infantile characteristic, not outgrown in ontogenetic evolution, which has been arrested in its march towards the summits of accuracy and precision of thought, popularly represented as truthfulness. What appears at first sight an unequivocal simulation will often be discovered to be in reality merely the product of a morbid fancy brooding over some imaginary situation, suggested perhaps by a sensational journal or some prurient acquaintance. The most familiar and striking, because the most easily understood example, is furnished by the periodic change of fashion in methods of suicide. Rigorous application of this very simple principle explains much of the seemingly intentional mendacious simulation of hysterics.

Several interesting cases were cited in support of this. One of a haunted house, taken from Grasset on "Spiritualism before Science," was particularly striking as a mixture of hallucinations and trickery. Another case of automatic writing with alleged revelations, taken from Esoaud de Messieres, was attributed to purely subliminal phenomena without trickery, and several accusations of rape were quoted in support of this contention. The distinctive characters of auto-accusation—apparent lucidity with a wealth of detail, the likeliness of the crime and the invariability of its recital—were supported by numerous cases varying from frank hallucinations to evident simulation.

The deterministic explanation of the mechanism of such cases, with its corollary of complete irresponsibility, was vehemently combated by Grasset, who adhered to the old doctrine of modified responsibility, which in judicial practice should not connote a lighter punishment. For him, the basis of responsibility is normality of the neurones, and as it is impossible to draw a fixed line separating mankind into two classes, entirely healthy and entirely morbid, it follows that there is a vast category of more or less inferior, though not totally irresponsible individuals, who yet cannot be held accountable to the full degree for their actions, and that it is the duty of the expert to appreciate to what degree they should be responsible to the law for such actions. But as criminals of this class cannot logically be placed in prison, and as they are not sufficiently insane to require detention in a lunatic asylum, it is urgently necessary to create special prison asylums. Society will thus be guarded against these half-insane people, while at the same time it is treating their deficiencies. The principle does not differ from that of isolation of cases of plague, small-pox or cholera. In this view Grasset was supported by Dupré and Régis. The latter urged that limitation of responsibility should not mean a shortened period of incarceration, since experience has shown that though a long term has sometimes benefited, a short sentence only aggravates a tendency to crime, so that the individual is not benefited, and society continues to suffer from his misconduct.

With regard to whether the simulations should be diagnosed as due to hysteria or to a fully conscious and voluntary effort, there is no certain criterion, for indeed, deliberate deception alternates with the work of unconscious fixed ideas. Williams urged, however, that in either case no deterrent is so powerful (and in this respect the hysteric does not differ from the normal), as a foreknowledge of the unpleasant results to oneself which an anti-social act entails, and to mitigate this unpleasantness is to loosen the social curb just with regard to the individuals to whom it is most necessary on account of the insufficiency of their own inhibitory power. The "impulsiveness" of the worse-than-savages who

terrorised Liverpool was restrained as soon as these scoundrels understood that a determined Chief of Police inflicted upon them the cat-o'-nine-tails; and such a stimulus differs only in degree from that exercised over the young girl who has not lost the power of preventing her hysterical attacks by the presence of some one who she knows will not hesitate to use such drastic measures as the "cold water cure." To lose sight of these principles is to deprive these unfortunates of their very best incentive to good conduct; for to pity and coddle is the very worst method of building a self-reliant character, and it is emphatically the lack of character and self-control that is the source of these patients' troubles. It is only when such social incentives fail to produce good conduct that mental alienation is constituted; and it follows that as society deprives itself of these incentives, in that proportion will mental alienation increase among such suggestible subjects. The "antiquated, common-sense" legislation in Great Britain has escaped the social danger of departing from this point of view, from which in France and America innumerable medico-legal subtleties have of late caused a divergence, the result of which is manifested by the enormous increase in crimes of violence committed by impulsive degenerates in those countries.

It was to the complication with mental degeneration that Dupré attributed most of the crimes of hysterics. Hysteria itself, in his view, eventuated rather in a fit than in an action foreign to the moral make-up of the patient. This is well illustrated by the fact that there is no authenticated case of a crime committed during hypnosis<sup>1</sup> at the instigation of the suggestioner. If the subject is urged to overstep the simulacrum of the act suggested, he responds by his typical fit. To this doctrine it may be objected that while a crime will not be committed out of harmony with the moral content, yet the extreme suggestibility of these subjects renders that moral content very susceptible to modification, and also renders an education towards criminality relatively easy. But at the same time it must not be forgotten, as Raymond has clearly pointed out in connection with the fugues, that every manifestation of an epileptic is not necessarily due to that condition; nor is the theft, the flight, or the arson of the psychasthenic necessarily a manifestation of that neurosis. Indeed, he and Janet have emphasised the fact that the fixed ideas of the psychasthenic do not eventuate in action, but, as in the case of Macbeth, they oscillate, "letting I dare not wait upon I would." Raymond distinguishes the hysterical impulsions by their secondary amnesia, which disappears if the second state returns, whereas they resemble the impulsions of degenerates in originating in fixed ideas, of which in the latter, however, the subject is always fully

<sup>1</sup> Babinski holds that hypnosis is merely a highly exaggerated hysterical state.

conscious. From the impulses of alcoholics it is very difficult to disassociate hysteria; for such neurotic subjects have generally a strong appetite for intoxicants, and are particularly susceptible to their influence, which often, indeed, determines temporary states of mental disequilibrium, which may eventuate in crime.

The character of the act, as in the preceding examples and in epilepsy and dementia præcox, generally permits one to detect the factor to which it is due, and hence to say how far it is due to disease. It is to this duty that the intervention of the physician should be confined, however difficult he may find it to so limit himself. He is called merely as a medical expert, and it is not his province to determine the fate of the patient from a social point of view; that is the duty of the Court, and the expert who allows his opinions to be coloured by considerations not belonging to his specialty arrogates to himself a function to which he has no right.

According to Leroy, actions which may be distinguished as hysterical are: (1) those which are due to the extreme suggestibility of the subject, which makes him the plaything of an influence such as a dream, a hallucination, or some outside agency; (2) those which are accomplished in the "second state," that is, where the everyday ego is unconscious of the act; (3) those which are due to instantaneous reaction to emotion, along with difficulty in stopping an action already commenced, the subject being for the time truly inaccessible to restraint exercised by himself or by others; (4) hysterical actions show in a marked way strange, romantic, mysterious, dramatic, sensational, bizarre characteristics, and are very often accompanied by naïveté and a crass want of foresight.

TOM A. WILLIAMS.

---

## Reviews

**ATLAS DER PATHOLOGISCHEN HISTOLOGIE DES NERVEN-SYSTEMS. III. Lieferung. Histologie des lésions expérimentelles et pathologiques des cellules nerveuses surtout des ganglions spinaux. V. BABES et G. MARINESCO. Berlin: Aug. Hirschwald. 1906.**

THIS fasciculus of the valuable Atlas edited by Dr Babes is devoted to the demonstration of new data regarding the fine structure of nerve-cells and the changes which their component elements undergo in various conditions of intoxication and infection, and

after injury to the axis-cylinder. It contains an introduction dealing with recent work regarding the fine structure of the nerve cell, and the relationship of the chromatic and the achromatic constituents to each other and to the neuro-fibrillæ. It contains also a brief *résumé* of the more important recent views regarding the distribution of the neuro-fibrillæ within the cell. It is to be regretted that the limitations of space have made this introduction somewhat too concise. Nine beautifully executed chromo-lithographic plates, containing sixty-four figures, show the fine changes in normal nerve-cells, in nerve-cells which have been poisoned by such substances as morphine, arsenic, snake venom, tetanus toxin and rabies, and also motor-cells and cells of the posterior root ganglia after section of nerves. The illustrations demonstrate with great clearness the remarkable changes which take place not only in the chromatic granules, but also in the neuro-fibrillæ, and in the nucleus and nucleolus. An indication is given of the reason for the hitherto insufficiently explained fact of the displacement and extrusion of the nucleus during the process of chromatolysis. The authors show that in certain cases, after section of the nerves, there is a peculiar localised swelling of the reticulum in part of the cell, which appears to be in such a position as to mechanically displace the nucleus towards the side of the cell.

As a demonstration of the recent work on the morbid changes in the nerve-cell, the figures leave little to be desired.

ALEXANDER BRUCE.

**THE DIAGNOSIS OF NERVOUS DISEASES.** PURVES STEWART, M.A., M.D., F.R.C.P. London: Edward Arnold. 1906. Price 15s.

WE have nothing but praise for this book, which it is our pleasant duty to review. The author has approached the diagnosis of nervous diseases from the clinical standpoint, "avoiding abstruse details of purely theoretical interest," and we heartily congratulate him upon the success which has attended his efforts. The two first chapters contain a clear and concise account of the chief anatomical and physiological points which are of importance to the clinician. The illustrations in these chapters are deserving of special mention; two diagrams which represent the most recent views as to the course of the motor and sensory tracts, are especially instructive. In the third chapter the author describes the method of case-taking which he is in the habit of using. Succeeding chapters deal with coma, fits, involuntary movements, aphasia, disorders of articulation, the cranial nerves, pain and other abnormal subjective sensations, abnormalities of sensation, organic motor paralysis,

recurrent and transient palsies, inco-ordination, postures and gaits, the trophoneuroses, reflexes, and affections of the sympathetic. In the last three chapters, hysteria, electro-diagnosis and electro-prognosis, and the cerebro-spinal fluid are considered. The excellence of the half-tone reproductions is a pleasing feature of the work. In conclusion, we may say that the book is admirably adapted to meet the wants of the physician who wishes to obtain in comparatively small compass an up-to-date knowledge of the diagnosis of nervous diseases which will prove of real service to him in practice. The book will also be read with interest and profit by those who have especially applied themselves to the study of the diseases of the nervous system.

EDWIN BRAMWELL

**OUTLINES OF COMPARATIVE LUNACY LAW.** (Beiträge zu einem Grundriss des vergleichenden Irrenrichtes.) Dr Jur. MARCUS WYLER, pp. 182. Halle a. S.: Carl Marhold. 1906.

AT a time like the present when various amendments to our lunacy laws are under consideration, such as the certification and detention of the victims of alcohol and drug habits, the insertion of clauses permitting the extra-asylum treatment of cases of incipient insanity, the formation of university clinics in psychiatry, and the establishment in England and Wales of the family-care system, a small pandect of the lunacy laws of all countries cannot but be of great value to medical men and others interested in these matters.

Dr Wyler is not a medical man, but is a jurist who has contributed many articles on this subject to various Continental journals, and, with the exception of Part III. of this book on the "State Supervision of the Insane," the whole of the present work has appeared in the *Psychiatrisch-Neurologische Wochenschrift* during the year 1905. Part I. deals with the "Legal Basis of the State Care of the Insane," and describes the essential features of the various legal enactments and stipulations concerning the insane in all European states and America. Wide divergences exist, and a perusal of this and the subsequent parts shows the force of what Dr Wyler pointed out in an able *précis* published in the *British Medical Journal* of 13th January 1906, that English law, by its confusion of poor law and lunacy law and its combination of public and private law, differs from that of almost every other country.

In Part II. the principal administrative forms as defined by law, the public and private asylums, and the family-care system,



are examined and compared. The whole matter is, of course, regarded solely under its legal aspect, and it seems possible that a certain ambiguity of law and laxity of procedure which the author deploras as present in certain countries is of positive advantage to the patients by permitting a greater freedom of medical treatment. In this connection it is of interest to note that in the author's judgment it is very doubtful whether certain states which have adopted the family-care system are *legally* justified in doing so. Dr Wyler's book differs from the distinguished work of Hermann Reuss, published in 1888, on the lunacy laws of Europe and North America, in that the latter described the various legal requirements in their geographical order, whereas Dr Wyler collates the information as to the several states under their proper categories, thus avoiding repetition and facilitating reference. The whole work, though small in bulk, must represent an immense amount of labour on the part of its author; and as it contains the essential features of the lunacy laws of Europe, and a complete list of references to the statutes at large of the several states, will be found indispensable to the student of comparative lunacy law.

R. CUNYNGHAM BROWN.

**A WALK THROUGH A MODERN ASYLUM.** (Ein Gang durch eine moderne Irrenanstalt.) By Dr H. HOPPE, of Königsberg. Pp. 75, with 16 plates. Halle a. S.: Carl Marhold. 1906. Price M. 1.60.

AFTER many years' service in public asylums, Dr Hoppe seeks in the present work to give an account of the inner life—*die Geheimnisse*—of a modern asylum. The book takes the form of a personally conducted tour through the Provincial Asylum of Galkhausen, between Cologne and Düsseldorf, and is particularly addressed to the lay public in the hope of allaying many fears and misconceptions about asylum treatment even to-day widely entertained. As, however, the construction and management of asylums in Prussia differs in many particulars from our own, this careful and intimate account of the Galkhausen Asylum will be found of much interest to both medical and lay readers in this country. The author opens by giving a brief historical account of the treatment—or rather, ill-treatment—extended to the unfortunate insane in the eighteenth and the beginning of the nineteenth centuries, ensuing upon the then current misconceptions of the nature of insanity, and the great changes which subsequently extended all over Europe, initiated by Conolly's abandonment of mechanical restraint.

From the administrative point of view the prominent features of the virtual revolution so brought about have been the careful classification of the patients according to the degree of liberty which might safely be permitted them, and, following upon this, the practical emancipation of great numbers of the insane. Although initiated by an Englishman, this movement for the granting of the greatest degree of liberty to the insane compatible with public safety has in Great Britain lagged far behind that of other countries, so that we have to-day only one institution in occupation—that at Kingseat near Aberdeen—at all corresponding to the village-asylums of Germany. Of these village-asylums Galkhausen affords a very good example, for the details of which readers are referred to Dr Hoppe's interesting description. The various detached houses which compose the institution are, as is usual, classified into closed, semi-closed, and open houses, and Dr Hoppe says that experience has taught the directors the error of timidity with regard to placing patients in the open houses. The beneficial influence of increased liberty is most marked, "the quarrelsome elements become peaceable sociable beings, the discontented grumbler ceases to complain, and the idler in restraint turns out as diligent a worker as the ordinary free man." Even in the closed houses, not only has mechanical restraint been entirely abolished, but the cellular system is hardly ever employed, rest in bed or the prolonged warm bath being substituted. With regard to the latter, the patients—presumably those who cannot endure the restraint of clothes—remain for hours, or days, and in some cases for weeks, day and night in the bath, almost invariably to the patient's great improvement. "Das Dauerbad," Dr Hoppe says, "bildet den besten Ersatz der früheren Tobzelle."

Dr Hoppe sorrowfully admits that the family-care system is not in practice at Galkhausen, not through any lack of recognition of its value, for this is, he says, undoubtedly the ideal form of the care of the insane, but simply because, and in this his opinion coincides with many others, the family-care system is best prosecuted in the form of autonomous colonies.

Dr Hoppe's *brochure* is well illustrated with photographs and plans, and ought not only to serve its purpose of reassuring the friends of insane persons as to the care experienced in modern asylums, but probably indicates the trend of asylum construction and management of the insane in other countries.

R. CUNYNGHAM BROWN.

# Bibliography

## ANATOMY

- KRONTHAL. Konstruktionsprinzipien des Nervensystems. (Schluss.) *Neurol. Centralbl.*, Nov. 1, 1906, S. 985.
- R. WEINBERG. Weitere Untersuchungen zur Anatomie der menschlichen Gehirnoberfläche. *Arch. f. Psychiat.*, Bd. 42, H. 1, 1906, S. 107.
- TROLARD. La circonvolution godronnée et ses prolongements sus-callosaux. *Rev. Neurol.*, oct. 30, 1906, p. 909.
- CLARENCE B. FARRER. Cerebral Topography at the Section Table. *Amer. Journ. Insan.*, Vol. lxiii, No. 1, 1906, p. 69.
- KOHLBRUGGE. Die Gehirnfurchen der Javenen. Eine vergleichend-anatomische Studie. Müller, Amsterdam, 1906, M. 7.
- TSUCHIDA. Ein Beitrag zur Anatomie der Sehstrahlungen beim Menschen. *Arch. f. Psychiat.*, Bd. 42, H. 1, 1906, S. 212.
- BUMKE. Ueber Variationen im Verlaufe der Pyramidenbahn. *Arch. f. Psychiat.*, Bd. 42, H. 1, 1906, S. 1.
- UGOLOTI. Sulle vie piramidali dell' uomo. *Riv. Speriment. di Freniatria*, Vol. xxxii, f. 3-4, 1906, p. 776.
- HELD. Zur Histogenese der Nervenleitung. *Anat. Anzeiger*, Bd. 29, Ergänzungsheft, 1906, S. 185.
- SCHULTZE. Nervenentwicklung und Zellstruktur. *Anat. Anzeiger*, Bd. 29, Ergänzungsheft, 1906, S. 285.
- VAN DER STRICHT. Sur la structure des cellules nerveuses. *Anat. Anzeiger*, Bd. 29, Ergänzungsheft, 1906, S. 286.
- VOGT. Fibrillenpräparate. *Anat. Anzeiger*, Bd. 29, Ergänzungsheft, 1906, S. 287.
- FRAGNITO. La prima apparizione delle neurofibrille nelle cellule spinali dei vertebrati. *Biblio. Anat.*, Bd. 15, f. 5, 1906, p. 290.
- BRUNO DA SILVA LOBO. Estrutura do cilindroeixo. *Arch. Brasil de Psychiat. e Neurol.*, Anno 2, N. 3, 1906, p. 213.
- EBNER. Ueber die Entwicklung der leimgebender Fibrillen im Zahnbein. *Anat. Anzeiger*, Bd. 29, Ergänzungsheft, 1906, S. 137.
- STAHR. Vergleichende Untersuchungen an den Geschmackspapillen der Orang-Utan-Zunge. *Ztschr. f. Morphol. u. Anthropol.*, Bd. 9, H. 3, 1906, S. 344.
- SCHULTZE. Zur Histogenese der peripheren Nerven. *Anat. Anzeiger*, Bd. 29, Ergänzungsheft, 1906, S. 179.

## PHYSIOLOGY

- CHARLES S. SHERRINGTON. The Integrative Action of the Nervous System. Archibald Constable, London, 1906, 16s.
- LEWANDOWSKY. Die Funktion des Centralnervensystem der Tiere und des Menschen. Fischer, Jena, 1906, M. 12.
- OSKAR VOGT. Ueber strukturelle Hirncentra, mit besonderer Berücksichtigung der strukturellen Felder des Cortex pallii. *Anat. Anzeiger*, Bd. 29, Ergänzungsheft, 1906, S. 74.
- EBSTEIN. Ein Beitrag zur Lokalisation an der Gehirnoberfläche. *Klinik f. psych. und nerv. Krankh.*, Bd. 1, H. 4, 1906, S. 273.
- MAXWELL. Chemical Stimulation of the Motor Areas of the Cerebral Hemispheres. *Journ. Biol. Chem.*, Oct. 1906, p. 183.
- LIEBEN. Zu Lehre von den Beziehungen der Grosshirnrinde zu den Pilomotern. *Zentralbl. f. Physiol.*, Okt. 20, 1906, S. 485.
- WEBER. Einwirkung der Grosshirnrinde auf Blutdruck und Organvolumen. *Arch. f. Anat. u. Physiol.*, H. 5-6, 1906, S. 495.
- SCHÜCKING. Sind Zellkern und Zellplasma selbständige Systeme? *Arch. f. Entwicklungsmechanik*, Nov. 6, 1906, S. 342.

- VON MIRAM. Ueber die Wirkung hoher Temperaturen auf den motorischen Froschnerven. *Arch. f. Anat. u. Physiol.*, H. 5-6, 1906, S. 533.
- ZWONITZKY. Ueber den Einfluss der peripheren Nerven auf die Warmerregulierung durch die Hautgefäße. *Arch. f. Anat. u. Physiol.*, H. 5-6, 1906, S. 465.
- WM. SUTHERLAND. The Molecular Theory of the Electric Properties of Nerve. *Amer. Journ. Physiol.*, Nov. 1, 1906, p. 297.
- BORUTTAN. Die Elektropathologie der Warmblüternerven sowie die Veränderungen der elektrischen Eigenschaften des Nerven überhaupt beim Absterben und Degenerieren. *Pflügers Arch. f. die ges. Physiol.*, Bd. 115, H. 5-6, 1906, S. 287.
- ENGELMANN. Zur Theorie der Contractilität. Reimer, Berlin, 1906, M. 1.
- ALBERTONI. Contribution à la connaissance de l'épuisement de l'activité de sens et de mouvement chez l'homme. *Arch. ital. de biol.*, Vol. xvi., 1906, p. 1.
- CLYDE BROOKS. On Conduction and Contraction in Skeletal Muscle in Water Rigor. *Amer. Journ. Physiol.*, Nov. 1, 1906, p. 218.
- ROUVIERE. Étude sur le développement phylogénique de certaines muscles sus-hyoidiens. *Journ. de l'anat.*, Nr. 5, 1906, p. 487.
- WM. SUTHERLAND. The Nature of Chemical and Electrical Stimulation. *Amer. Journ. Physiol.*, Nov. 1, 1906, p. 266.
- GEMITZ und WINTERSTEIN. Ueber die Wirkung erhöhter Temperatur auf die Reflexerregbarkeit des Froschrückenmarks. *Pflügers Arch. f. die ges. Physiol.*, Bd. 115, H. 3-4, 1906, S. 273.
- LUSSANA. La funzione dei canali semi-circolari. *Riv. Speriment. di Freniatria*, Vol. xxxii., f. 3-4, 1906, p. 577.
- KITAGAWA und THIERFELDEN. Ueber das Cerebrum. III. Mitteil. *Hoppe-Seylers Ztschr. f. physiol. Chemie*, Bd. 49, H. 2-3, 1906, S. 286.
- PANELLA. Le nucléone et l'eau du cerveau chez les animaux à jeun. *Arch. ital. de biol.*, Vol. xvi., 1906, p. 145.
- BURNETT. The Influence of Temperature upon the Contraction of Striped Muscle and its Relation to Chemical Reaction Velocity. *Journ. Biol. Chem.* Oct. 1906, p. 195.
- MÖBIUS. Ueber den physiologischen Schwachsinn des Weibes. Marhold, Halle, 1907, M. 1.50.

### PSYCHOLOGY

- DUPUREUX. L'application de la recherche des tests mentaux de Binet chez les enfants des écoles communales de Gand. *Journ. de Neurol.*, nov. 5, 1906, p. 555.
- MEYER. Rausch und Zurechnungsfähigkeit. *Arch. f. Psychiat.*, Bd. 42, H. 1, 1906, S. 163.
- LAPPONI. Hypnotismus und Spiritismus. Elischer, Leipzig, 1906, M. 4.
- DEXLER. Das Scheuen der Pferde, Stampede of Horses, Tierpaniken. *Arch. f. Psychiat.*, Bd. 42, H. 1, 1906, S. 194.
- KATZ. Versuche über den Einfluss der "Gedächtnisfarben" auf die Wahrnehmungen des Gedächtnisses. *Zentralbl. f. Physiol.*, Nov. 3, 1906, S. 517.
- PAUL PROVOTELLE. Françoise Fontaine, possédée de Louviers (1591). *Ann. méd.-psychol.*, nov.-déc. 1906, p. 353.
- A. BABEL. Anomalies observées dans les rapports sociaux. *Ann. méd.-psychol.*, nov.-déc. 1906, p. 369.
- ZIEHEN. Erkenntnistheoretische Auseinandersetzungen. *Ztschr. f. Psychol. u. Physiol. der Sinnesorgane*, Bd. 43, H. 4, 1906, S. 241.
- HANS ABELS. Ueber Nachempfindungen im Gebiete des kinästhetischen und statischen Sinnes. *Ztschr. f. Psychol. u. Physiol. der Sinnesorgane*, Bd. 43, H. 4, 1906, S. 268.

### PATHOLOGY

- PURVES STEWART and JULIUS BERNSTEIN. A Case of Partial Doubling of the Spinal Cord. *Rev. Neurol. and Psychiat.*, Nov. 1906, p. 729.
- HIRSCHWALD. Zur Pathogenese des Basedow'schen Symptomenkomplexes. *Zentralbl. f. Nervenheilk. u. Psychiat.*, Nov. 1, 1906, S. 833.
- KAUFFMANN. Zur Pathologie des Stoffwechsels bei Myasthenie. *Zentralbl. f. d. ges. Physiol. u. Pathol. des Stoffwechsels*, Nr. 19, 1906, S. 593.
- EIMIGER. Beiträge zur Kenntnis der Gefäßveränderungen in der Gehirnrinde bei Psychosen. *Arch. f. Psychiat.*, Bd. 42, H. 1, 1906, S. 161.

- MEDEA. Contributo allo studio delle fini alterazioni della fibra nervosa (fenomeni de- e rigenerativi) nella neurite parenchimatosa degenerativa sperimentale. (Cont. e fine.) *Riv. Speriment. di Freniatria*, Vol. xxxii., f. 3-4, 1906, p. 899.
- BALLI. Lesioni del reticolo neurofibrillare endocellulare in mammiferi adulti totalmente o parzialmente privati dell'apparecchio tiro-paratiroideo e loro rapporto colla temperatura. *Riv. Speriment. di Freniatria*, Vol. xxxii., f. 3-4, 1906, p. 803.
- SCARPINI. Sulle alterazioni delle cellule nervose dell'ipertermia sperimentale studiate con i metodi di Donaggio. *Riv. Speriment. di Freniatria*, Vol. xxxii., f. 3-4, 1906, p. 725.
- GOUREWITCH. Contribution à l'étude de la résistance du réseau fibrillaire des cellules nerveuses de la moelle épinière des lapins adultes. *Riv. Speriment. di Freniatria*, Vol. xxxii., f. 3-4, 1906, p. 928.
- HASSLAUER. Die Mikroorganismen bei den endokraniellen otogenen Komplikationen. *Centralbl. f. Ohrenheilk.*, Bd. 5, H. 1, 1906, S. 1.
- KUTSCHER. Ein Beitrag zur Agglutination der Meningococcen. *Deutsche med. Wchnschr.*, Nov. 15, 1906, S. 1849.
- GOSIO. Circa il reperto fenolico nelle culture di taluni aspergilli e penicilli. *Riv. Speriment. di Freniatria*, Vol. xxxii., f. 3-4, 1906, p. 920.
- MARINESCO. Du rôle des cellules apotrophiques dans la régénérescence nerveuse. *Comptes rend. de la Soc. de Biol.*, nov. 16, 1906, p. 381.
- MARINESCO et MINEA. Précocité des phénomènes de régénérescence des nerfs après leur section. *Comptes rend. de la Soc. de Biol.*, nov. 15, 1906, p. 383.
- VENEZIANI. Colorazioni positive delle fibre nervose degenerate, nel nervo tentacolare di *Helix pomatia*. *Biblio. Anat.*, Bd. 15, f. 5, 1906, p. 259.
- DEGANELLO. Dégénérescences dans le névraxe de la grenouille consécutives à l'exportation du labyrinthe de l'oreille. *Arch. ital. de biol.*, Vol. xvi., 190, p. 156.

## CLINICAL NEUROLOGY AND PSYCHIATRY

### GENERAL—

- EICHHORST. Pathologie und Therapie der Nervenkrankheiten. Urban und Schwarzenberg, Wien, 1907, M. 9.
- WM. CAMPBELL POSEY and WM. G. SPILLER. The Eye and Nervous System; their Diagnostic Relations by Various Authors. Lippincott Company, Phila. and London, 1906, 25s.
- MEYER. Compendium der Neurologie und Psychiatrie. Speyer und Kaerner, Freiburg, 1907, M. 3.60.
- FOREL. L'Âme et le Système nerveux. Steinheil, Paris, 1906, 5 fr.
- HÜBNER. Zur Lehre von der Lues nervosa. *Berl. klin. Wchnschr.*, Nov. 5, 1906, S. 1448.
- OPPENHEIM. Nervenkrankheit und Lektüre; Nervenleiden und Erziehung. die ersten Nervosität des Kindesalters. Zweite Auflage. S. Karger, Berlin, 1907, M. 2.
- GAUPP. Der Einfluss der deutschen Unfallgesetzgebung auf den Verlauf des Nerven- und Geisteskrankheiten. *Münch. med. Wchnschr.*, Nov. 13, 1906, S. 2233.

### SPINAL CORD—

- Tabes.**—WAYNCOP. Crises gastriques au début du tabes et crises gastriques en dehors du tabes. (*Thèse.*) Roussel, Paris, 1906.
- FRENKEL. L'ataxie tabétique. Ses origines, son traitement par la rééducation des mouvements. Félix Alcan, Paris, 1906, 8 fr.
- Myelitis.**—EBSTEIN. Myelitis acuta (post influenzam?), Heilung. *Klinik f. psych. u. nerv. Krankh.*, Bd. 1, H. 4, 1906, S. 278.
- Fracture Dislocation.**—BEVERLEY WELFORD. Fracture-Dislocation of the Spine. *Brit. Med. Journ.*, Nov. 10, 1906, p. 1270.
- Disseminated Sclerosis.**—AUSTREGESILLO e GOTUZZO. Tres casos atypicos de esclerose em placas. *Arch. Brasil. de Psychiat. e Neurol.*, Anno 2, N. 2, 1906, p. 127.
- Syringomyelia.**—GRAMEGNA. La radioterapia della siringomielia. Nota di tecnica sulla radioterapia del midollo spinale. *Riv. crit. di clin. med.*, Nov. 10, 1906, p. 717.
- Landry's Paralysis.**—WHARTON SINKLER. Case of Landry's Paralysis with Recovery. *Journ. Nerv. and Ment. Dis.*, Nov. 1906, p. 692.

- Lumbar Puncture.**—MORELLI. Esame del liquido cefalo-rachidiano. Valore diagnostico e prognostico. *Clinica Mod.*, Ott. 24, 1906, p. 505.  
**MINOT.** Le Diagnostic précoce de la syphilis nerveuse par la ponction lombaire. (*Thèse.*) Storck et Cie, Lyon, 1906.

**BRAIN—**

- Meningitis.**—RIEBOLD. Ueber seröse Meningitis. *Deutsche med. Wchnschr.*, Nov. 15, 1906, S. 1859.  
**LINDEMANN.** Sind die Steinkohlengruben die Verbreiter der Genickstarre? *Münch. med. Wchnschr.*, Okt. 30, 1906, S. 2160.  
**Hemiplegia.**—F. H. EDGEWORTH. On Transitory Hemiplegia in Elderly Persons. *Scot. Med. and Surg. Journ.*, Nov. 1906, p. 414.  
**Tumour.**—T. GRAINGER STEWART. The Diagnosis and Localisation of Tumours of the Frontal Regions of the Brain. *Lancet*, Nov. 3, 1906, p. 1209.  
**MORTON PRINCE.** Limited Area of Anaesthesia, Epileptiform Attacks of Hemialgesia, and Early Muscular Atrophy in a Case of Brain Tumour. *Journ. Nerv. and Ment. Dis.*, Nov. 1906, p. 698.  
**HARVEY CUSHING.** Sexual Infantilism with Optic Atrophy in Cases of Tumour affecting the Hypophysis Cerebri. *Journ. Nerv. and Ment. Dis.*, Nov. 1906, p. 704.  
**HUGH E. JONES and THURSTAN HOLLAND.** Demonstration of an Exostosis of the Frontal Sinus, and a Skiagraph of the same. *Brit. Med. Journ.*, Nov. 17, 1906, p. 1367.  
**Abcess.**—PERCY GOLDSMITH. Some Unusual Cases of Frontal Sinus Suppuration. *Brit. Med. Journ.*, Nov. 17, 1906, p. 1369.  
**DUFAYS.** Accidents cérébraux graves au cours d'une mastoïdite chronique réchauffée. *Rev. hebdom. de Laryngol.*, Nov. 17, 1906, p. 580.  
**SCHROEDER.** Ein weiterer Fall von otogener eitriger Sinusphlebitis mit fieberlosen Verlauf. *Ztschr. f. Ohrenheilk.*, Bd. 51, H. 4, 1906, S. 357.  
**Sinus Thrombosis.**—WIMMER. Ein Fall von ausgedehnter Thrombosierung der Hirnsinus. *Berl. klin. Wchnschr.*, Nov. 12, 1906, S. 1475.  
**Bulbar Paralysis.**—OSANN. Ueber Bulbärparalyse bei Lipomatose. *Arch. f. Psychiat.*, Bd. 42, H. 1, 1906, S. 180.  
**FÜNCI NAKA.** Eine seltene Erkrankung der Pyramidenbahn mit spastischer Spinalparalyse und Bulbärsymptomen. *Arch. f. Psychiat.*, Bd. 42, H. 1, 1906, S. 19.  
 **Amaurotic Family Idiocy.**—KARL SCHAFFER. Beiträge zur Nosographie und Histopathologie der amaurotisch-paralytischen Idiotieformen. *Arch. f. Psychiat.*, Bd. 42, H. 1, 1906, S. 127.  
**General Paralysis.**—VALLET. Contribution à l'étude des remissions dans la paralysie générale. (*Thèse.*) Michalon, Paris, 1906, fr. 2.50.  
**BOUCHAUD.** Un cas de main de prédicateur chez un paralytique général. *Rev. Neurol.*, oct. 30, 1906, p. 917.  
**KARL LIEBSCHER.** Die cytologische und chemische Untersuchung des Liquor cerebrospinalis bei Geisteskrankheiten, insonderheit bei progressive Paralyse. *Wien. med. Wchnschr.*, Nr. 45, 1906, S. 2210.  
**WASSERMANN.** Ueber das Vorhandensein syphilitischer Antistoffe in der Cerebrospinalflüssigkeit von Paralytikern. *Deutsche med. Wchnschr.*, Nov. 1, 1906, S. 1769.  
**Cerebellum.**—HÖNCK. Ueber die Rolle des Sympathicus bei der Erkrankung des Wurmfortsatzes. Fischer, Jena, 1906, M. 4.

**ALCOHOL, ETC.**

- BIANCHI.** L'alcool e la malattia del sistema nervoso. *Il Tommasi*, Anno 1, No. 23, 1906.  
**ROBERT JONES.** Mental Degradation the Result of Alcohol. *Amer. Journ. Insan.*, Vol. lxiii., No. 1, 1906, p. 39.  
**MAURICIO DE MEDEIROS.** Notas de um anti-alcoolista. *Arch. Brasil. de Psychiat. e Neurol.*, Anno 2, N. 2, 1906, p. 149.

**MENTAL DISEASES—**

- R. S. WOODWORTH.** Psychiatry and Experimental Psychology. *Amer. Journ. Insan.*, Vol. lxiii., No. 1, 1906, p. 27.  
**CLARENCE B. FARKER.** Clinical Psychiatry: Clinical Demonstrations. *Amer. Journ. Insan.*, Vol. lxiii., No. 1, 1906, p. 75.

- Lunacy in London. Editorial. *Brit. Med. Journ.*, Nov. 10, 1906, p. 1311.
- KOVALEVSKY. Type mongol de l'idiotie. *Ann. méd.-psychol.*, nov.-déc. 1906, p. 431.
- VOGT. Die mongoloide Idiotie. *Klinik f. psych. u. nerv. Krankh.*, Bd. 1, H. 4, 1906, S. 347.
- LAQUER. Die ärztliche und erziehlliche Behandlung von Schwachsinnigen (Debilien und Imbezillen) in Schulen und Anstalten und ihre weitere Versorgung. (Fortsetz.) *Klinik f. psych. u. nerv. Krankh.*, Bd. 1, H. 4, 1906, S. 320.
- ALAN RIGDEN. Presidential Address concerning the Insanity of Child-birth. *Brit. Med. Journ.*, Nov. 10, 1906, p. 1253.
- BOSC. Diabète et troubles mentaux. *Rev. de Psychiat.*, oct. 1906, p. 416.
- MOREIRA et PEIXOTO. Les maladies mentales dans les climats tropicaux. *Arch. Brasil. de Psychiat. e Neurol.*, Anno 2, N. 3, 1906, p. 222.
- HENRIQUE ROXO. Perturbacoes mentaes ligadas á arterio-sclerose. *Arch. Brasil. de Psychiat. e Neurol.*, Anno 2, N. 3, 1906, p. 197.
- FINCKH. Die psychischen Symptome bei Lues. *Centralbl. f. Nervenheilk. u. Psychiat.*, Nov. 15, 1906, S. 865.
- CHARLES RICKSHER. A Review of the Mental Symptoms accompanying Apoplexy. *Amer. Journ. Insan.*, Vol. lxiii, No. 1, 1906, p. 55.
- DE CLÉRAMBAULT. Sur un cas de délire collectif où figure un paralytique général. *Ann. méd.-psychol.*, nov.-déc. 1906, p. 378.
- RODIET et CANS. Diagnostic différentiel des troubles cérébraux d'origine toxique dus à l'alcool et au tabac et de la paralysie générale, d'après les symptômes oculaires. *Ann. méd.-psychol.*, nov.-déc. 1906, p. 408.
- PIGHINI. Il ricambio organico nella demenza precoce. *Riv. Speriment. di Freniatria*, Vol. xxxii, f. 3-4, 1906, p. 513.
- FAUSER. Zur Kenntnis der Melancholia. *Centralbl. f. Nervenheilk. u. Psychiat.*, Nov. 15, 1906, S. 880.
- BODROS. De la prétendue démence des persécutés. (Thèse.) Michalon, Paris, 1906, fr. 1.75.
- SCHAIKEWICZ. Ueber Geisteskrankheiten im russischen Heer während des russisch-japanischen Krieges. *Centralbl. f. Nervenheilk. u. Psychiat.*, Nov. 15, 1906, S. 872.
- STIEDA. Ueber Geisteskrankheiten im russischen Heer während des russisch-japanischen Krieges. *Centralbl. f. Nervenheilk. u. Psychiat.*, Nov. 15, 1906, S. 875.
- LUGIATO e CHANNESSIAN. La pressione sanguigna nei malati di mente. *Riv. Speriment. di Freniatria*, Vol. xxxii, f. 3-4, 1906, p. 737.
- IBBA. Citolisine termolabili e coctostabili nel sangue dei paicopatici. *Riv. Speriment. di Freniatria*, Vol. xxxii, f. 3-4, 1906, p. 642.
- PODESTA. Häufigkeit und Ursachen der Selbstmordneigung in der Marine im Vergleich mit der Armee. *Arch. f. Psychiat.*, Bd. 42, H. 1, 1906, S. 32.
- SIGWART. Selbstmordversuch während der Geburt. *Arch. f. Psychiat.*, Bd. 42, H. 1, 1906, S. 249.
- GRASSET. Les Devoirs et les Droits de la Société vis-à-vis des aliénés. Blais et Roy, Paris, 1906.
- PIGHINI. La criminalità negli stadi iniziali della "demenza precoce." *Riv. Speriment. di Freniatria*, Vol. xxxii, f. 3-4, 1906, p. 859.
- SCHNITZER. Moderne Behandlung der Geisteskranken. Herm. Walther, Berlin, 1906, M. —50.
- HOCQUET. Des sorties provisoires à titre d'essai. Contribution à la thérapeutique des maladies mentales. (Thèse.) Michalon, Paris, 1906, fr. 2.50.
- GUSTAVO ARMBRUST. O tratamento physico da obesidade. *Arch. Brasil. de Psychiat. e Neurol.*, Anno 2, N. 2, 1906, p. 167.
- GRASSET. La psychothérapie totale ou supérieur. *Rev. de Psychiat.*, oct. 1906, p. 397.
- PERPÈRE. Deux asiles de Saint-Petersbourg. *Rev. de Psychiat.*, oct. 1906, p. 412.
- ADAM. Des établissements d'aliénés, d'idioti et d'épileptiques. Du rôle du médecin dans ces établissements. (Suite et fin.) *Ann. méd.-psychol.*, nov.-déc. 1906, p. 421.

#### GENERAL AND FUNCTIONAL DISEASES—

- Epilepsy.**—WEICHARDT und PILTZ. Experimentelle Studien in die Eklampsie. *Deutsche med. Wchnschr.*, Nov. 15, 1906, S. 1854.
- CENI. Nuove ricerche sulla natura dei principi tossici contenuti nel siero di sangue degli epilettici. *Riv. Speriment. di Freniatria*, Vol. xxxii, f. 3-4, 1906, p. 451.

- SALA. Sull' anatomia patologica dell' epilessia. *Riv. Speriment. di Freniatria*, Vol. xxxii., f. 3-4, 1906, p. 482.
- RICCI. Studio critico sopra 393 casi di epilessia. (Cont. e fine.) *Riv. Speriment. di Freniatria*, Vol. xxxii., f. 3-4, 1906, p. 813.
- BESTA. Ricerche sopra la pressione sanguigna il polso e la temperatura degli epilettici. (Cont. e fine.) *Riv. Speriment. di Freniatria*, Vol. xxxii., f. 3-4, 1906, p. 460.
- BESTA. Significato e frequenza delle manifestazioni emilaterali nell' epilessia essenziale. *Riv. Speriment. di Freniatria*, Vol. xxxii., f. 3-4, 1906, p. 665.
- HEINRICH STADELMANN. Cerebrale Kinderlähmung und Epilepsie. *Wien. med. Wchnschr.*, Nr. 45, 1906, S. 2206.
- BERNHARDT. Beitrag zur Lehre vom Status hemiepilepticus. *Berl. klin. Wchnschr.*, Nov. 5, 1906, S. 1443.
- HOPPE. Die Beziehungen der Bromwirkung zum Stoffwechsel der Epileptiker. *Neurol. Centralbl.*, Nov. 1, 1906, S. 993.
- Neurasthenia.**—STEKEL. Die Ursachen der Nervosität. Knepler, Wien, 1906, M. 1.
- GERHARDT. Die Differentialdiagnose der nervösen Herzstörungen. *Klinik f. psych. u. nerv. Krankh.*, Bd. 1, H. 4, 1906, S. 298.
- FRANÇAIS. L'apepsie dans les névroses. *Arch. gén. de méd.*, oct. 30, 1906, p. 2761.
- Hysteria.**—HENRIQUE ROXO. Hysteria. *Arch. Brasil. de Psychiat. e Neurol.*, Anno 2, N. 2, 1906, p. 139.
- ROUBY. Lourdes und die Hysterie. Frankfurter Verlag, 1906, M. —50.
- INGEGNIEROS. Le langage musical et ses troubles hystériques. Félix Alcan, Paris, 1906, 6 fr.
- GOLDFLAM. Ein Fall von hysterischem Fieber. *Neurol. Centralbl.*, Nov. 1, 1906, S. 978.
- Tetany.**—MARCO ALMAGIÀ. Sul rapporto tra sostanza nervosa centrale e tossina del tetano. *Sperimentale*, Anno 60, f. 5, 1906, p. 654.
- Traumatic Neurasthenia.**—ALESSI. Nevrosi traumatiche in un arteriosclerotico. *Clinica mod.*, oct. 17, 1906, p. 493.
- GIGLIOLI. Alcune considerazioni a proposito delle traumato-nevrosi. *Riv. crit. di clin. med.*, Nov. 10, 1906, p. 722.
- Angio-neuroses.**—ERNEST DORE. On Cutaneous Affections in Various Diseases, with especial Reference to Certain Angio-neuroses. *Brit. Journ. of Dermatol.*, Oct. 1906, p. 354.

#### SPECIAL SENSES AND CRANIAL NERVES—

- ONODI. Beiträge zur Lehre der durch Erkrankung der hintersten Siebbeinzelle und der Keilbeinhöhle bedingten Sehstörung und Erblindung. *Berl. klin. Wchnschr.*, Nov. 19, 1906, S. 1514.
- BOUCHAUD. Un cas d'ophtalmoplégie unilatérale, totale et complète avec cécité du même côté. *Journ. de Neurol.*, nov. 5, 1906, p. 549.
- BLOCH. Ueber willkürliche Erweiterung der Pupillen. *Deutsche med. Wchnschr.*, Nov. 1, 1906, S. 1777.
- KUTNER. Abnorme Erschöpfbarkeit der Lichtreaktion der Pupille (Asthenische Lichtstarre). *Centralbl. f. Nervenheilk. u. Psychiat.*, Nov. 1, 1906, S. 825.
- BÉRIEL. Un cas de paralysie faciale obstétricale spontanée. *Rev. mens. des mal. de l'enfance*, nov. 1906, p. 503.
- JULIUS DONATH. Die Sensibilitätsstörungen bei peripheren Gesichtslähmungen. *Neurol. Centralbl.*, Nov. 16, 1906, S. 1039.
- MOSSI. Otite scléreuse bilatérale; surdité, vertiges. Traitement par les ponctions lombaires. *Rev. heb. de Laryngol.*, Nov. 17, 1906, p. 593.
- FERDINANDO MASSEI. Ueber die Bedeutung der "Anästhesie des Kehlkopf-eingangs" bei den Recurrenslähmungen. *Berl. klin. Wchnschr.*, Nov. 19, 1906, S. 1512.
- ROSENBACH. Gibt es bis jetzt eine Ausnahme von der Regel, dass bei intensiver Affection der Nn. recurrentes vagi die Abduktoren der Stimmbänder früher Funktionsstörungen zeigen als die Adduktoren. *Berl. klin. Wchnschr.*, Nov. 12, 1906, S. 1480.
- DEGE. Zur Aetiologie der Lähmung des Nervus laryngeus inferior. *Berl. klin. Wchnschr.*, Nov. 5, 1906, S. 1446.
- GEORGE L. RICHARDS. Two Cases of Abductor Paralysis. *Brit. Med. Journ.*, Nov. 17, 1906, p. 1874.



## MISCELLANEOUS SYMPTOMS—

- HENRY SMURTHWAITE. Headache: Pathological Conditions of the Middle Turbinal a Causal Factor. *Brit. Med. Journ.*, Nov. 17, 1906, p. 1368.  
 HUGO LUKACS. Spasmus progrediens (Torticollis mentalis). *Centralbl. f. Nervenheilk. u. Psychiat.*, Nov. 1, 1906, S. 829.  
 MICHAEL LAPINSKY. Die Zustand der Reflexe in paralysierten Körpertheilen bei totaler Durchtrennung des Rückenmarkes. *Arch. f. Psychiat.*, Bd. 42, H. 1, 1906, S. 55.  
 WALTON. The Cerebral Element in the Reflexes and its Relation to the Spinal Element. *Journ. Nerv. and Ment. Dis.*, Nov. 1906, p. 681.  
 SOUQUES. Aphasie motrice sans lésion de la troisième circonvolution frontale. *Bull. et Mém. de la Soc. méd. des Hôp. de Paris*, oct. 19, 1906.  
 VON MAYENDORF. Ueber eine direkte Leitung vom optischen zum kinästhetischen Rindenzentrum der Wort- und Buchstabenbilder. *Wien. klin. Wchnschr.*, Nov. 8, 1906, S. 1335.  
 VON MONAKOW. Aphasie und Diachisis. *Neurol. Centralbl.*, Nov. 16, 1906, S. 1026.

## TREATMENT\*—

- EBSTEIN. Einige Bemerkungen zur Behandlung der syphilitischen Erkrankungen des Nervensystems. *Klinik f. psych. u. nerv. Krankh.*, Bd. 1, H. 4, 1906, S. 234.  
 MULTAVIDI. Die Macht des Hypnotismus im Dienste des Menschen. Verlag Meteor, Dresden, 1906, M. 2.  
 FEDOR KRAUSE. Die operative Behandlung der Hirn- und Rückenmarkstumoren. *Wien. med. Presse*, Nr. 46, 1906, S. 2376.  
 LAVAL. Beiträge zur operativen Freilegung des Bulbus venae jugularis. *Arch. f. Ohrenheilk.*, Bd. 69, H. 3-4, 1906, S. 161.

\* A number of references to papers on Treatment are included in the Bibliography under the individual Diseases.

## BOOKS AND PAMPHLETS RECEIVED.

- Eichhorst, Hermann. Pathologie und Therapie der Nervenkrankheiten. Urban und Schwarzenberg, Berlin.  
 Magelssen. Norway as a Winter and Summer Health Resort. Kristiania, 1906.  
 Posey and Spiller. The Eye and Nervous System. J. B. Lippincott Co., Philadelphia and London, 1906.  
 Wilson. Anatomy of the Calamus Region in the Human Bulb. *Journ. Anat. and Physiol.*, 1906.  
 Schiefferdecker. Neurone und Neuronenbahnen. Barth, Leipzig.  
 Determann. Physikalische Therapie der Erkrankungen des Zentralnervensystems, inklusive der allgemein Neurosen. F. Enke, Stuttgart, 1906.  
 Babinski. Ma conception de l'Hystérie et de l'Hypnotisme. Durane, Chartres, 1906.  
 Somner, Robert. Klinik für psychische und nervöse Krankheiten, Bd. 1, H. 4. Marhold, Halle, 1906.  
 Möbius. Über Scheffel's Krankheit. Marhold, Halle, 1906.  
 Lewandowsky. Die Funktionen des Zentralnervensystems. Fischer, Jena, 1907.  
 Mott, Haliburton, and Edmunds. Regeneration of Nerves. *Proc. Roy. Soc.*, Vol. 78, 1906.  
 Mott. The Microscopic Changes in the Nervous System in a Case of Chronic Dourine or Mal de Coit, and Comparison of the Same with those found in Sleeping Sickness. *Proc. Roy. Soc.*, Vol. 78, 1906.  
 Mott. Alcohol and Insanity. Adlard & Son, London, 1906.  
 Oppenheim. Nervenkrankheit und Lektüre. Nervenleiden und Erziehung. S. Karger, Berlin, 1907.  
 Sherrington. The Integrative Action of the Nervous System. Charles Scribner's Sons, New York, 1906.  
 Arch. Brasil. de Psychiat., Neurol. e Sciencias Affins, Nos. 2 e 3, 1906. Rio de Janeiro.

# Indices

*Page references to Original Articles are indicated by heavy type figures.*

## SUBJECT INDEX.

- ABSCCESS**; *vide* Brain  
**Abducens**: Paralysis of, in Otitis, 765  
**Accommodation**; *vide* Pupil  
**Achilles-Jerk**; *vide* Reflexes  
**Acromegaly**: Spinal Cord, Degeneration in, 429; with Lesion of Hypophysis and Sella Turcica, 759  
**Acropathies**: Intense Cold in Pathogeny of, 155  
**Afferent Nervous System** from New Aspect, 47  
**Alcohol**: Ocular Symptoms due to, 837; Injections in Neuralgias, 236; in Contractures, Spasms, and Tremors of Limbs, 710; *vide* Neuritis  
**Alcoholism**: Want of Insight in, 589; Mixed Conditions in Epilepsy and, 589; Psychic and Motor Disturbances in, 591; Crimes committed during Intoxication, 592, 593 (2); Atypical Alcoholic Psychoses, 705; Hallucinatory Dementia in, 705; Pseudo-Paralysis of, 705; Delirium Tremens, 231  
**Alexia**: Developmental, 518  
**Amaurotic Family Idiocy**, 568, 570, 571  
**Amnesia**: Retro-antegrade in Hysteria, 223  
**Amphioxus**: Cranial and Spinal Ganglia and Viscero-motor Roots in, 419  
**Amyotrophic Lateral Sclerosis**, 750; Pathology of, 638  
**Anæsthesia**, Limited Area of, in Brain Tumour, 835  
**Aneurisms**: Intracranial, 507; Aortic, and Tabes, 565  
**Aphasia**: Word-Blindness, 152, 518; Treatment of Visual, 152; Aphasia in Migraine, 223; and Mental Disease, 304; Lesions of Left First Temporal Convolution and Sensory Aphasia, 329; Sensory Aphasia, with Right Homonymous Hemianopia, 377; Tactile, 586, 703; Revision of Question, Third Left Frontal Convolution does not play Special Rôle, 649; Localisation and Pathological Physiology of Sensory, 650; Clinical and Pathological Study of, 651; Agrammatismus and Disturbance of Internal Language, 652; Hysterical Dysarthria, 654; Alexia, 152, 518; Subcortical (Pure), 845; *vide* Apraxia, Amnesia  
**Apoplexy**: Cerebellar, 506  
**Apraxia**: in General Paralysis, 306; Mixed, 380  
**Arsenical Neuritis**, 216  
**Arteries**: Hypertonus in Sclerosed, 362; Hæmorrhage from Middle Meningeal, 364; Hæmorrhage into Brain and Cord from Obliterative Disease of, 407  
**Arterio-Sclerosis**: Mental Diseases associated with Cerebral, 458; Manic-Depressive Insanity and, 707; Traumatic Neurasthenia in, 761  
**Ascending Paralysis**, 828; Cord and Medulla in, 106; *vide* also Landry's Paralysis  
**Asthenias and Myopathic Atrophies**, 295  
**Asynergy**: Cerebellar, and Inertia, 703  
**Ataxia**: in Childhood, 297; Sensory, 446  
**Athetosis**, Double, 146; Nerve Transplantation in, 386  
**Atrophy**: Myopathic, and Asthenias, 295; Experimental Cerebral and Cranial Atrophies, 742; of Muscles in Tumours of Brain, 835  
**Autogenic Regeneration**; *vide* Regeneration  
**Axis-Cylinder**; *vide* Nerve  
**BABINSKI Sign**; *vide* Reflexes  
**Baræsthesia**, Disturbances of, 446  
**Basedow's Disease**; *vide* Exophthalmic Goitre  
**Beri-Beri**, Polyneuritic Psychoses and, 848  
**Birds** after Section of Posterior Spinal Roots, 821  
**Bladder**, Functions of, in Hemiplegia, 152; Nerves of, in Cat, 275  
**Blindness**: of Cortical Origin, from

- Double Hemianopia, 699; Anatomy of Cortical, 372; *vide* Hemianopia
- Blood, in Epilepsy, 286
- Blood Pressure and Neurasthenia, 58
- "Blue Disease," 54
- Brain: *Anatomy*: The Claustrum, 31; Histology of Cerebellum, 32; Width of Cortex in Estimation of Development, 33; Cerebral Cortex of Dolphin, 34; Hind-Brain of Pig, 126; Histology of Cerebral Cortex, 387; the Tænia Pontis, 413; Caput Gyri Hippocampi, 414; Radix Mesencephalica Trigemina and Ganglion Isthmi, 413; Olfactory Bulband Cornu Ammonis, 417; Estimation of Skull Capacity on Cadaver, 420; Nervus Intermedius of Wrisberg and Bulbo-Pontine Gustatory Nucleus, 473; Neuroglia Framework of Cerebellum, 683; Lobus Cerebelli Medianus, 737; *vide* Nerve Tracts
- Physiology*: Respiratory Centre in Cerebral Cortex of Dog, 35; Central Respiratory Innervation, 36; Experimental Section of Pyramids in Dogs and Apes, 207; Experimental Destruction of Hypophysis, 207; Separate Sensory Centres in Parietal Lobe for Limbs, 296; Edinger - Westphal Nucleus, 288; Organic Changes in Development of Association, 278; Localisation of Cerebral Function, 308; Cholesterin of Brain (2), 351; Function of Left Prefrontal Lobe, 362; Cortical Visual Area and Macula Lutea, 372; Projection of Retina on Cortex of Occipital Lobe, 372; Localisation of Higher Psychic Functions, and Prefrontal Lobe, 420; Motor Areas in Cerebral Cortex of *Dasyurus Viverrinus*, 635; Functions of Caudate Nucleus, 817; Cortical Representation of Cutaneous and Muscular Sensibility, 819
- Pathology*: Medulla in Acute Ascending Paralysis, 106; Malformation of Brain in *Hatteria Punctata*, 214; Heterotopia of Cortical Substance, 428; Cerebellum in General Paralysis, 561; Brain of Microcephalic Idiot, showing lack of Corpus Callosum, 639; Senile Brain, 753
- Clinical*: Minor Aids in Examination of Brain Disease, 52; Miliary Disseminated Brain Syphilis, 572; Symptoms of Frontal Disease, 646; Pontine Hæmorrhage, 754; Lesion of Uncinate Region of Temporo-Sphenoidal Lobe, 833; Ocular Symptoms in Toxic Disturbances due to Alcohol and Tobacco, 837; *vide* Encephalitis, Tumours, Cerebellum
- Brain Surgery: Present Status of, 852
- Bulbar Paralysis: Pseudo-Bulbar Palsy in Child, 584
- Bulbar Symptoms from Intoxication, 300; Thermo-Asymmetry, 62
- CAISSON Disease, 755
- Catatonica: 226
- Cauda Equina, Diseases of, 292
- Caudate Nucleus, Functions of, 817
- Cerebellum: Histology of, 32; Neuroglia Framework of, 683; Lobus Cerebelli Medianus, 737; Asynergy and Inertia in Disease of, 703; Atrophy of, 139; Hæmorrhage, 506; in General Paralysis, 561; Idiocy in Cerebellar Lesion, 523; Position of Head in Disease of, 455; *vide* Tumours, Atrophy of Cells of Purkinje, 55.
- Cerebral; *vide* Brain
- Cerebro-Spinal Fluid: Choline in, 285; Clinical Significance of, 504; Relation of Syphilis to Lymphocytosis of, 749; Lumbar Puncture in Psychiatry and Neurology, 655; Lymphocytosis in Juvenile General Paralysis, 813
- Cholesterin of Brain (2), 351
- Choline in Cerebro-Spinal Fluid, 285
- Chorea: its Neuronic Aspect, 218; Tetanoid, Associated with Cirrhosis of Liver, 249; of Childhood, Treatment of, 382; Osteitis Deformans with Huntingdon's, 840
- Cirrhosis of Liver, Association with Tetanoid Chorea, 249
- Claudication; *vide* Intermittent Claudication
- Confabulation: Psychology of, 227; Clinical Significance of, 228
- Consciousness: Non-Epileptic Affections of, 575
- Contractures in Organic Nervous Diseases, 375
- Conus terminalis: Diseases of, 292; Pathology of Epiconus Medullaris, 567; Traumatic Lesions of, 567
- Convulsions in Typhoid, 143; in Children and Relation to Epilepsy, 839
- Corpus Callosum: Lack of in Brain of Microcephalic Idiot, 639; Tumour of, 694
- Cortex; *vide* Brain
- Cortical Blindness: Anatomy of, 372
- Cretinism: Familial, 224
- Crural Monoplegia: 296
- Cysticercus Cellulosæ in the Insane, 373
- DEAFNESS due to Hysteria, etc., 516
- Degeneration: of Nerve Tissue, 637; Retrograde, in Spinal Nerves, 558, 686, 747

- Delirium: produced by Toxines of Pellagra, 45; by Drugs, 83  
 Delirium Tremens, 231  
 Dementia Paralytica; *vide* General Paralysis  
 Dementia: Pathological Anatomy, 382; Hallucinatory, in Alcoholics, 705  
 Dementia Præcox: 302, 519; Certain Forms of Psychosis allied to, 287; Hebephrenic Forms of, 707; Heboid Paranoid Group of, 708; Mild Forms of, 848  
 Dendrites and Diseases, 636  
 Development of Cranial and Spinal Nerves of Human Embryo, 30  
 Diabetes: and Pneumococcal Cerebro-Spinal Meningitis, 752  
 Digestive Organs: Head's Zones in Disorders of, 701  
 Diphtheria: Precocious Paralysis of Palate in, 608; Paralysis of Tongue, 217  
 Disseminated Sclerosis: 139, 502; in Guise of Transverse Myelitis, 51; Frequency of Certain Signs and Symptoms, 601; Mental Disorders in, 642; Diagnosis from Psychogenic Neuroses, 831  
 Disseminated Syphilitic Encephalitis, 644  
 Dissociated Personality: in Hysteria, 840  
 Diver's Palsy, 755  
 Donaggio's Method: Changes in Nerve Cell with, 587  
 EAR Disease, Psychical Importance of, 776  
 Eclampsia: Puerperal, and Parathyroid Insufficiency, 577  
 Edinger-Westphal Nucleus, Physiological Significance of, 288  
 Encephalitis: Cerebral Symptoms due to, 140; Relation to Acute Anterior Poliomyelitis, 140; Disseminated Syphilitic, 644  
 Enteric Fever: Convulsions in, 143; Abdominal Reflex in, 582; Philopovicz's Sign in, 378; Recurrent Third Nerve Paralysis in, 454; Abnormal Reflex in, 582  
 Epiconus Medullaris; *vide* Conus  
 Epilepsy: Etiology of, 574; Pathology of, 354; Pathology of Epileptic Idiocy, 689; Blood in, 288; Borderland of, 696; in course of Chronic Psychoses, 381; Vertigo in, 694; with Unilateral Manifestations, 696; Variations of Mood in, 523; Cessation of Pulse during Onset of Fits, 579; Peculiar Attitudes during Sleep, 839; and Migraine, 839; Mixed Conditions and Alcoholism, 589; Reflex Epilepsy, 838; Relation to Convulsions in Children, 839; Jackson and Pseudo-Jacksonian, 768; Significance of Jacksonian, 511, 647; Paramyoclonus Epilepticus, 19; Non-epileptic Affections of Consciousness, 575; Diet in, 235, 383; Bleeding in, 383; Potassium Bromide in, 852; Treatment by Appendicostomy, 579  
 Ergography, Laws of, 40, 128, 280, 282; Rest in Ergographic Work, 283; Mechanical Value of Mental Representation, 283; Effect of Standing Position on, 283; Effect of Preliminary Immobility on, 283; Economy of Effort, 283; Effect of Slackening of Rhythm, 283; Influence of Orientation on Activity, 283  
 Exhaustion from Excess of Function, 424  
 Exophthalmic Goitre: Parathyroid Glands in, 46; Pathogenesis of, 59; Heart Neuroses and, 697; Antithyroid in Treatment of, 65; Serum Treatment of (2), 384, 445; Surgical Treatment of, 462, 711; Pathology of, 647; Treatment with Röntgen Rays, 711  
 Eyes: Paralysis of Associated Movements—Voluntary and Automatic-Reflex Movements, 454  
 FACE: Hemiatrophy of, 1; Tic of, 56, 220; Peripheral Hemispasm of, 57; Synergic Paradoxical Contractions following Palsy of, 57; Bilateral Circumscribed Atrophy, 221; Facial Paralysis and Hemiatrophy of Tongue, 289; Peripheral Facial Palsy, 690  
 Fatigue: Laws of Ergography, 40, 128, 280, 282; Fatigue Curves in Diagnosis of Nervous Diseases, 224; Mental Fatigue in School Children, 285; *vide* Ergography  
 Fever: Hysterical (2), 443  
 Formic Acid: Muscular-Tonic Action of, 232  
 Freud's Method of Treatment in Hysteria, 462  
 Freidreich's Ataxia, 217, 441  
 Frontal Lobes; *vide* Brain  
 GALL-BLADDER, Contractile Mechanism and Extrinsic Nervous Control, 37  
 Ganglia: Lesions of Gasserian and Posterior Root Ganglia and Herpes, 139; Cranial and Spinal in Amphioxus, 419; Excision of Gasserian in Facial Neuralgia, 793; Extirpation of Gasserian, 795  
 Gasserian, *vide* Ganglia  
 General Paralysis: Pathology of, 73,

- 169, 266, 537, 616; Histopathological Changes of Cerebellum in, 561; Fibrillar Structure in, 212; with Miliary Disseminated Brain Syphilis, 572; Etiology and Duration of, 645; Trauma and, 229, 307, 813; Tabes, Syphilis, and, 356; Supposed Immunity of Syphilitic Arabs to, 513; Somatic Evidences of Syphilis in, 512, 748; Ocular Symptoms in, 837; Apraxia in, 306; Pupil Accommodation Reflex in, 307; Multiple Lipomata in, 369; Perforating Ulcer in, 369; Red-Sores in, 370; Cerebro-spinal Lymphocytosis in Juvenile, 813; Early Diagnosis and Treatment of, 847; Juvenile General Paralysis, 813
- German Measles: Polyneuritis as Sequela of, 356
- Glands: Parathyroid Insufficiency in Eclampsia, 577; Atrophy of, in Syphilis, 583
- Graves's Disease, *vide* Exophthalmic Goitre
- HABIT Spasm in Children, 145
- Hallucinations of Peripheral Origin, 776
- Head-Nodding and Nystagmus in Infancy, 771
- Hæmorrhage: Surgical Intervention for Intra-cranial, 66; Spinal, 137; from Middle Meningeal Artery, 364; into Brain and Cord from Obliterative Arterial Disease, 407; Post-Traumatic, from Superior Longitudinal Sinus, 443
- Hæmatemesis in Organic Nervous Diseases, 154
- Head: Position of, in Cerebellar Disease, 455
- Head Injuries: 463; Indications for Operative Treatment (2), 463
- Head's Zones: in Disorders of Digestive Organs, 701
- Heart-Neuroses and Basedow's Disease, 697
- Hemialgesia: Epileptiform Attacks of, in Brain Tumour, 835
- Hemianæsthesia in Migraine, 223
- Hemianopia: Retrogression of, after Paralytic Attacks, 148; Right Homonymous Lateral, and Sensory Aphasia, 377; Blindness of Cortical Origin from, 699; Bi-Temporal, 699
- Hemiatrophy: of Face, 1; of Tongue, 289
- Hemicraniosis, 61
- Hemiparesis in Migraine, 223
- Hemiplegia; following Whooping-Cough, 54; Infantile Cerebral, 146; Functions of Bladder in Cerebral, 152; Chronic Progressive Double, 182; in Vascular Lesions, 365; Adult, 366: Sensory Disturbances in Cerebral, 367; Post-Hemiplegic Movements, 368; Transitory, 362, 505, 833; Infantile Cerebral, 506
- Hemispasm: Peripheral Post-Paralytic, 56; Peripheral Facial, 57
- Herpes Zoster: Lesions of Gasserian and Posterior Root Ganglia in, 139; Kernig's Sign in, 300
- Heterotopia of Cerebral Cortical Substance, 428
- Huntingdon's Chorea with Osteitis Deformans, 840
- Hydrotherapy in Mental Diseases, 777
- Hyperæsthesia of Visual Periphery, 580
- Hypertonus in Sclerosed Arteries: Cerebral Manifestations of, 362
- Hypnosis: What is it? 445
- Hypoglossus: Diphtheritic Paralysis of, 217
- Hypophysis Cerebri, *vide* Pituitary Body
- Hysteria: Traumatic, resembling Ophthalmoplegia Externa, 144; Hysterical Tic, 144; Retro-antegrade Amnesia in, 223; Symptoms and Associations of, 293; Stigmata caused by Organic Brain Lesions, 297; Hysterical Fever (2), 443; Immobility of Pupil in, 453; Treatment of, at Massachusetts General Hospital, 462; Freud's Method of Treatment by "Psycho-Analysis," 462; "Hysteria" of Animals, 513; Deafness due to, 516; Hysteria Analysis, 522; Hysterical Dysarthria, 654: "Twilight-State," 760; Responsibility of Hysterics, 760; "Stammering-Gait," 770; Hystero-melancholia, 774; From Point of View of Dissociated Personality, 840
- IDIOCY: 225; Forms which result from Meningitis, 156; Amaurotic Family, 568, 570, 571; Pathology of Epileptic, 689; In Cerebellar Lesion, 523
- Impulses: Pathogenesis of Some, 459
- Infantile Cerebral Hemiplegia, Disturbances of Movement in, 146
- Infantile Paralysis: 440; Porencephalic Form of, 571
- Infantilism with Optic Atrophy in Tumour of Hypophysis Cerebri, 835
- Innervation: Central Respiratory, 36
- Insanity, *vide* Mental Diseases and Psychiatry
- Instinct, 686
- Intermedio-lateral Tract, Nerve Cell of, 349
- Intermittent Claudication: 153; 698; of Spinal Cord, 584; 842; of Nervous Centres, 585

- Insanity:** *Cysticercus Cellulosæ* in, 372 ; Mental Processes in Retardation and Excitation of, 303 ; Common Form of, 457 ; *vide* Mental Diseases
- Insular Sclerosis,** *vide* Disseminated Sclerosis
- Ischæmia:** Muscular Atrophy in, 514 ; Sensation in Acute, 151
- JAW-MOVEMENTS:** Physiology of, 558
- Juvenile Dementia,** 456
- KERNIG'S Sign:** in Herpes Zoster, 300
- Knee Angle Phenomenon** in Tabes, 501
- Knee-Jerk,** *vide* Reflexes
- Korsakow's Symptom-Complex:** in Brain Syphilis, 704
- LANDRY'S Paralysis:** 106 ; 829 ; after Enteric, 291, *vide* also Ascending Neuritis
- Larynx:** Structure and Function of Taste-buds, 43 ; in Tabes, 290 ; Laryngeal Crises in Tabes, 692 ; Zur Recurrens Frage, 769
- Laughter,** Spasmodic, 371
- Light Perception,** Peculiar Alteration in, 449
- Lille, Report of Congress at:** 713 ; 797 ; 868
- Lipomata** in General Paralysis, 369
- Localisation:** of Cerebral Function, 308 ; of Cortical Visual Area, 372 ; of Macula Lutea, 372 ; in Nuclei of Cranial and Spinal Nerves, 416 ; of Higher Psychic Functions, 420 ; of Musical Talent in Brain and on Skull, 586
- Lumbar Puncture:** in Psychiatry and Neurology, 655 ; *vide* Cerebro-spinal Fluid
- Lunacy Law,** 869, *vide* Mental Diseases
- MACROPSIA and Micrographia,** 449
- Macula Lutea,** 372
- Manic-Depressive Insanity,** 849
- Measles:** Transverse Myelitis as Sequela to, 751
- Megrim,** *vide* Migraine
- Memory:** Experimental Observations on, 305
- Meningeal Artery:** Hæmorrhage from, 364
- "Meningeal Irritation,"** 749
- Meningitis:** *Diplococcus Intracellularis* Meningitidis and Gonococci, 740 ; Experimental, 826 ; Meningococcal Pharyngitis in Epidemic Cerebro-spinal, 354 ; Epidemic Cerebro-spinal in India, 361 ; Diabetes and Pneumococcal, 752 ; Poliomyelitis Anterior Acute and, 830 ; Idiocy resulting from, 156 ; Treatment of, 234 ; Serum Treatment of, 826 ; Operative Treatment of, 791
- Mental Diseases:** Pathological Anatomy of, 287, 387 ; Toxic Cause in some Forms of, 640 ; Pathogenesis of some Impulses, 459 ; Types in, 455 ; Mental Symptoms in Cerebellar Tumour, 218 ; Idiocy and Cerebellar Lesion, 523 ; Mental Symptoms associated with Cerebral Arterio-sclerosis, 458, 707 ; Mental Symptoms in Multiple Sclerosis, 642 ; Epilepsy in Course of Chronic Psychoses, 381 ; Slightly Abnormal Children, 380 ; Amentia, 772 ; Acute Juvenile Deterioration, 225 ; Psychoses of Involution Period and Juvenile Dementia, 456 ; The Dementias, 383 ; Depressions of Advanced Life, 159 ; Manic Depressive Insanity, 849 ; Hysterio-melancholia, 774 ; Coming of Psychasthenia, 518 ; Mental Confusion, 379 ; Circumscribed Autopsychosis on Basis of a Morbidly Dominant Idea, 461 ; Atypical Alcoholic Psychoses, 705 ; Polyneuritic Psychoses, 848 ; Relation of Aphasia and, 304 ; Insight in, 379 ; Mixed Apraxia, 380 ; Affectivity, Suggestibility, Paranoia, 383 ; "Zwangsvoränge," 460 ; Feeling of Unreality, 519 ; Pseudæsthesia, 768 ; Primary Incoherence with Excitement, 775 ; Hallucinations of Peripheral Origin, 776 ; False Reminiscence, 847 ; Diagnostic Association Studies, 520 ; Psychical Importance of Ear Disease, 776 ; Clinic for (Review), 238 ; Forensic Import of Sexual Perversity, 850 ; Discharge of Patients from Asylums in Prussia, 851 ; Outlines of Comparative Lunacy Law (Review), 869 ; Treatment, 63, 64 ; of Acute Insanity in General Hospitals, 709 ; Hydrotherapy in, 777 ; Walk through a Modern Asylum (Review), 870 ; *vide* General Paralysis, Dementia Præcox, Psychiatry, Psychoses.
- Methods:** of Staining Neuroglia, 205 ; Pseudo-vital Method, 417 ; Ramon y Cajal's Silver Method applied to Axis-Cylinders, 420 ; Changes in Nerve Cells by Donaggio's Method, 557 ; Pyridine Methods for Differentiation of Reticulum of Nerve Elements, 684 ; Weigert's Neuroglia Stain, 738
- Method of Inscribing Tracings on Revolving Cylinder,** 208
- Microcephalic Idiot:** Brain of, 639
- Micrographia:** Relation to Macropsia, 449

- Migraine:** Aphasia, Hemiparesis and Hemianæsthesia in, 223; and Epilepsy, 839
- Monoplegia, Crural,** 296
- Multiple Sclerosis, *vide* Disseminated Sclerosis**
- Muscles:** Activity in Skeletal Muscle-fibre, 127; Function of Degenerate Muscles, 214; Tonic Action of Formic Acid, 232; Reaction of Striated Muscle to Nicotine and Curari, 276; Paralysis of, Involuntary, 39, 279; Functioning of Degenerate, 559
- Muscular Atrophy:** Ischemic, contractions and Paralysis, 514; Neuritic Type of Progressive, 691; in Brain Tumour, 835
- Muscular Dystrophy affecting Hands and Feet,** 192; *vide* Myopathy
- Musical Talent:** Localisation of, 586
- Myasthenia Gravis,** 146, 295
- Myelitis:** Transverse, simulating Multiple Sclerosis, 51; Pathology of Myelitis Acutissima Hemorrhagica Disseminata, 401; Descending Degenerations of Posterior Columns in Transverse, 468; Extension of Ascending, 741; Transverse, as Sequela of Measles, 751
- Myoclonus:** Multiplex, 143; Epilepticus, 19
- Myopathy:** Primary Progressive, in two Brothers, 563; Asthenias and Myopathic Atrophies, 296; Muscular Dystrophy affecting Hands and Feet, 192
- NARCOLEPTIC Attacks,** 575
- Nerves:** Decussation of Trochlear Nerve, 203; Wrisberg's Intermediary Nerve, 473; Distribution of Afferent Nerves in Skin, 738; Localisation of Nuclei of Cranial and Spinal, 416; Motor Nuclei after Lesions of Peripheral, 288; Zur "Recurrentfrage," 769; Motor Nerve Endings in Reptiles, 202; Cranial Nerve Components in Petromyzon, 418; to Cat's Bladder, 275; Trophic Nerves, 437; Consequences of Injury to Peripheral, 47; Mechanism of Regeneration, 134; Autogenic Regeneration, 209; Retrograde Degeneration in Spinal, 686, 747; Wallerian Law, 210; Influence of Facial Hemiatrophy on, 1; Distribution and Recovery of Peripheral, 430; Paralysis of External Branch of Spinal Accessory in Tabes, 439; Rheumatic Polyneuritis in Cranial, 690; Paralysis of Abducens in Otitis, 765; Recurrent Paralysis of Third Nerve in Typhoid, 454; Diphtheritic Paralysis of Hypoglossus, 217; Local Action of Cocaine and Stovaine on Peripheral, 828; Neurotropism and Transplantation of, 745; Surgery of Peripheral, 860
- Nerve Cells:** Structure of, 416, 556; Cell types in Sensory Ganglia, 124; Modifications by Nissl's Method, 123; Alterations by Donaggio's Method, 557; Metabolism and Action of, 277; Reproduction of, 820; Reaction to Certain Poisons, 276; Atrophy of Cells of Purkinje, 55; Morbid Conditions of Endocellular Fibrillar Reticulum, 557, 687, 739; Reaction to Fasting and Cold, 687
- Nerve Fibres:** Reticulated Structure of Axis Cylinder, 737; Chromatic Pseudocorpuscles of Axis-cylinder, 123; White Rami Fibres and Spinal Accessory Nerve, 33; *vide* Regeneration and Degeneration.
- Nerve Fibrils:** Fibrillar Structure in Progressive Paralysis, 212; Post-mortem Changes in Neuro-fibrils, 352; Club-like Terminals in Collateral Regeneration, 825
- Nerve Palsies:** Nerve Transplantation in, 886
- Nerve Roots:** Viscero-motor, in Amphioxus, 419; Effect on Peripheral Neurons of Section of Posterior, 743; Movements of Birds after Section of Posterior Spinal Roots, 821; Tabetic Lesions of Spinal, 825
- Nerve Tracts:** Cranial and Spinal Nerves in Occipital Region, 30; Inferior Longitudinal Bundle and Central Optic Bundle, 31; Faisceau en Crochet, ou Faisceau Cérébello-Bulbaire, 203; Sensory Conduction in Cord, 205; Paralysis by Compression of Pyramidal, 693
- Nerve Transplantation in Nerve Palsies and Athetosis,** 386
- Nervous Diseases:** Diagnosis of (Review), 868
- Nervous System:** Action of Snake Venoms on, 498; Resuscitation of Central, 741; Technique of Operations on Central, 778; Atlas of Pathological Histology of (Review), 867
- Nervousness:** Psychological Conception of, 370
- Neuralgia:** Treatment by Alcoholic Injections, 236; Facial, treated by Excision of Gasserian Ganglion, 793, 795; Surgical Treatment of Trigeminal, 857
- Neurasthenia:** and Blood-Pressure, 58; Traumatic, in Arterio-Sclerotics, 761

- Neuritis, possibly due to working with Artificial Manures, 50; Arsenical, 216; Alcoholic, 564; Rheumatic Polyneuritis of Cranial Nerves, 690; Neuritic Type of Progressive Muscular Atrophy, 691; Peripheral Neuritis in Tabes, 692; Ascending and Chronic Rheumatism, 828
- Neuroglia: Framework of Cerebellum, 688; Methods of Staining, 205, 738
- Neuroses: Food Factor in Paroxysmal, 236; Psycho-Physical Methods in Traumatic, 587; Heart-Neuroses and Basedow's Disease, 697; Diagnosis between Multiple Sclerosis and Psychogenic, 881
- Neurotropism and Transplantation of Nerves, 745
- Nuclei: of Cranial and Spinal Nerves, 416; after Lesions or Peripheral Motor Nerves, 288
- Nystagmus: and Head-Nodding in Infancy, 771
- OCCIPITAL Lobes, *vide* Brain
- Ocular Movements: Isolated Palsy for Lateral Movement of Internal Rectus Muscle, 375; Paralysis of Movement Upwards and Downwards, 763; Paralysis of Associated Movements, 454; Paralysis of Abducens in Otitis, 765; *vide* Ophthalmoplegia
- Ocular Symptoms: in Toxic Cerebral Disturbances and in General Paralysis, 837
- Oculo-motor Nerve, Recurrent Paralysis of in Typhoid, 454
- Ophthalmoplegia: Hysteria with Clinical Picture of External, 144; Bilateral, 302; Interna unilaterale, 841
- Optic Disc: Congestion of, with Spindle-Shapes Enlargement of Blind Spot, 301
- Optic Tracts: Inferior Longitudinal Bundle and Central Optic Bundle, 31
- Osmotic Theory of Sleep, 423
- Osteitis Deformans: with Huntingdon's Chorea, 840
- Otitis: Paralysis of Abducens in, 765
- PAIN: Sense of, 149; Meaning and Mechanism of Visceral, 700; Head's Zones in Digestive Disorders, 701; Projection of Sensation from Lower to Upper Extremity, 702
- Palate: Precocious Paralysis of, in Diphtheria, 608; Undescribed Symptom in Paralysis of, 221
- Paradoxical Contractions following Palsy of Face, 57
- Paramyoclonus Epilepticus, 19
- Paralysis: Cord and Medulla in Acute Ascending, 106; Anatomy of Syphilitic Spinal, 211; Undescribed Symptom of Palatal, 221; Cortical Tactile, 299; Surgical Methods in, 384; Infantile, 440; Isolated Traumatic, 499; "Crutch" Paralysis, 564; by Compression of Pyramidal Tract, 693; of Pneumonia in Adults, 767
- Paralysis Agitans: Pathological Anatomy of, 689
- Paraplegia: Lacunar and Myelopathic, 53; from Retraction in Old People, 566; Cutaneous and Tendon Reflexes in Spastic, 700
- Parathyroid Glands in Graves's Disease, 46
- Parietal Lobes, *vide* Brain
- Parkinson's Disease, *vide* Paralysis Agitans
- Paroxysmal Neuroses, Food Factor in, 236
- Pellagra: Delirium produced by Toxines of, 45
- Peroneal Form of Muscular Atrophy, 829
- Petromyzon: Cranial Nerve Components of, 418
- Pharyngitis, Meningococcal, in Epidemic Cerebro-Spinal Meningitis, 354
- Philipovitch's Sign in Enteric Fever, 378
- Pituitary Body: Experimental Destruction of, 207; Tumour of, 429; Tumour of with Infantilism and Optic Atrophy, 835
- Pneumonia: Paralyzes of, 767; Paralyzes induced by Pneumococcal Infection, 44; Pneumococcal Cerebro Spinal Meningitis, 752
- Poliomyelitis Anterior Acute: Bacteriology of, 132; Relation to Encephalitis, 140; Rhizopods in, 353; and Cerebro Spinal Meningitis, 830
- Poliomyelitis Anterior Chronica, 137
- Polynervitis: as Sequela of German Measles, 356; Toxic, in Phthisical Patient, 500; Rheumatica in Cranial Nerves, 690; Psychoses in, 448; *vide* Neuritis
- Porencephaly, 571
- Post-Hemiplegic Movements, 368
- Pott's Paraplegia, 218, 359, 752
- Pressure, Sense of, 446
- Progressive Muscular Atrophy, Neural form of, 691, 829
- Pseudo-Sclerosis: Westphal's, 831
- Pseudo-Systemic Sclerosis of Cord, 139
- Psychasthenia, 518
- Psychiatry: Observations on Case of Murder and Suicide, 231; Lumbar



- Puncture in, 655 ; Position of the Atypical Child, 656 ; *vide* Mental Diseases
- Psychic Functis ; on Localisation of Higher, 420
- Psychological Conception of Nervousness, 370
- Psychological Methods : Disclosure of Fact by, 305
- Psychology : Scientific Investigation of Psychical Processes in Higher Animals, 822 ; of Sudden Religious Conversion, 457 ; Psycho-Analogies and Association Experiments, 520
- Psychoses : Epilepsy in Chronic, 381 ; of Children, 461 ; of Involution Period, 456 ; Puerperal, 588 ; Types of Devolutional, 665 ; *vide* Mental Diseases
- Pulse : Cessation of, during onset of Epileptic Fits, 579
- Pupil : Psychical and Sensory Reaction of, 149 ; Accommodation Reflex in General Paralysis, 307 ; Traumatic Immobility of (3), 451 ; Traumatic Reflex Immobility of, 452 ; Immobility of in Hysteria, 453 ; Reflexes in Mitral Valve Lesions, 581
- Purkinje, Atrophy of Cells of, 55
- Pyramidal Tracts : Latent Forms of Affections of, 54 ; Unilateral Ascending Paralysis due to Degeneration of, 296 ; Experimental Section in Dogs and Apes, 207 ; Paralysis by Compression of, 693
- Pyridine method for Differentiation of Reticulum of Nerve Elements, 684
- RAMON Y CAJAL's Method applied to Axis-cylinder, 420
- "Recurrents Frage," 769
- Recurrent Third Nerve Paralysis in Typhoid, 454
- Reflex Epilepsy, 838
- Reflexes : Cerebral and Spinal Elements in, 843 ; Babinski's Sign in Scarlet Fever, 151 ; "Extensor Phenomenon," 152 ; Pharyngeal, 154 ; Abdominal, in Enteric, 222 ; Dorsal Foot, 447 ; Peculiar Reflex in Affection of Central Motor Neurones, 448 ; Schäfer's Antagonistic, 448 ; in Infantile Cerebral Hemiplegia, 506 ; Unilateral Loss and Subsequent Return of Knee-Jerk, 515 ; Abdominal Reflex in Enteric, 582 ; in Spastic Paraplegia, 700 ; Abdominal Reflex in Frontal Tumours, 809 ; New Method of Investigating Knee and Ankle-Jerks, 844 ; Simple Device for obtaining Knee-Jerk, 844
- Regeneration : in Peripheral Nerves, 184, 209, 210, 425, 430, 558, 637
- Reptiles, Nerve-Endings in, 202
- Respiration : Cortical Centre in Dog, 35 ; Central Respiratory Innervation, 36
- Retina : Projection on Cortex of Occipital Lobe, 372 ; Hyperæsthesia of Visual Periphery, 580
- Retinal Cyanosis, 54
- Reviews : Die Erkrankungen des Rückenmarkes und der Medulla Oblongata, 162 ; Klinik für psychische und nervöse Krankheiten, 238 ; Gehirn und Rückenmark, 239 ; Die Temperamente, 240 ; Localisation of Cerebral Function, 308 ; Histology of Cerebral Cortex, 387 ; Affectivität, Suggestibilität, Paranoia, 393 ; Palpablen Gebilde des normalen menschlichen Körpers, 465 ; Robert Schumann's Disease, 526 ; Atlas der pathologischen Histologie des Nerven Systems, 867 ; Diagnosis of Nervous Diseases, 868 ; Outlines of Comparative Lunacy Law, 869 ; Walk through a Modern Asylum, 870 ; Journal of Abnormal Psychology, 527 ; Gehirn und Seele, 528 ; Psychotherapeutische Briefe, 657
- Rheumatism : Nervous Manifestations of Acute, 59 ; Polyneuritis of Cranial Nerves, 690 ; and Ascending Neuritis, 828
- SCARLET Fever : Babinski Sign in, 151
- Sciatica : Surgical Treatment of, 465
- Sclerosis : Cerebral, 427 ; *vide* Disseminated Sclerosis
- Sensation : Thermo-Asymmetry of Bulbar Origin, 62 ; in Acute Localised Ischaemia, 151 ; Rare Form of Pseudo-Æsthesia, 768 ; Disturbances of Baræsthesia, 446 ; Representation in Cortex, 819
- Sensory Ataxia, 446
- Sensory Areas in Disorders of Digestion, 701
- Serum Reaction in Persons suffering from Infective Conditions, 562
- Serum Treatment : in Exophthalmic Goitre (2), 384 ; of Cerebro-spinal Meningitis, 826
- Sexual Perversity, Forensic import of 850
- Skin : Distribution of Afferent Nerves in, 738
- Skull : Estimation of Capacity of, 420
- Sleep : Osmotic Theory of, 423 ; Peculiar Attitude in Epilepsy during, 839
- "Sleep Drunkenness" : 229
- Sleeping Sickness, 113

- Snake Venom, Action on Nervous System, 498
- Spasmodic Laughter and Weeping, 371
- Sphenoidal Sinusitis, Cerebral and Ophthalmic Complications in, 757
- Spinal Accessory Nerve: White rami F bres and, 33; Paralysis of, in Tabes, 419
- Spinal Cord: *Anatomy*: Structure of in Ostrich, 34; Recognition of Segmental Levels from Appearance of Transverse Section, 344; Distribution of Cells in Intermedio-Lateral Tract, 349; Lumbo-Sacral-Coccygeal Cord in Macaque Monkey, 683; Cell Columns of Anterior Cornua, 737
- Spinal Cord: *Physiology*: Sensory Conduction in, 205; Paths for Painful and Thermal Impressions, 424
- Spinal Cord: *Pathology*: Partial Doubling of, 6, 729; in Acute Ascending Paralysis, 106; Degeneration in Acromegaly, 429; Descending Degenerations of Posterior Columns in Transverse Myelitis and Compression of Dorsal Posterior Roots by Tumours, 428; Malformation of, 497; Extirpation of Lower Half of, 562;
- Spinal Cord: *Clinical*: Lesions from Absorption from Localised Septic Foci, 25; *vide* Conus Terminalis and Various Diseases of
- Spondylitis Deformans, 361
- Suprarenal Capsules in Nervous and other Diseases, 352
- Syphilis: Tabes, General Paralysis and, 556, 512, 561, 748; Miliary Disseminated Brain Syphilis and General Paralysis, 572; Atrophy of Glands at Base of Tongue in, 583; Korsakow's Symptom-Complex in Brain, 704; Relation to Lymphocytosis of Cerebro-Spinal Fluid, 749; Syphilitic Spinal Paralysis, 211
- Syringobulbia, 368; 832
- Syringomyelia: with Double Optic Neuritis, 138; with Sensory Disturbances of Radicular Distribution, 442; Extending from Sacral Region to Upper Part of Right Internal Capsule, 832
- TABES: 433; Juvenile, 136; Hæmamestis in, 154; Late, 289; Larynx in, 290; General Paralysis, Syphilis and, 356; Forms Seldom Described, 358; Dystrophy of, 437; Paralysis of Spinal Accessory in, 439; and Knee Angle Phenomenon, 501; Surgical Complications of, 501; and Aortic Aneurism, 565; Ocular Crises in, 642; Chancre in, 692; Peripheral Neuritis and Arthropathy of Knee in, 692; Laryngeal Crises in, 692; Co-existence of Tertiary Syphilitic Lesions with, 748; Lesions of Spinal Nerve Roots, 825; Pal's teaching as to Vascular Crises in, 830
- Tactile Paralysis, 299
- Taste-buds of Larynx, 43
- Temperaments (Review), 240
- Tendon-Jerks, *vide* Reflexes
- Tendons: Indications for Transplantation of, 237; Failure in Transplantation of, 862
- Tetany: 762; Prognosis of Adult, 698
- Tetanoloid Chorea, 249
- Thalamic Syndrome, 648
- Thalamus, a co-ordinating Centre, 368
- Thermo-Asymmetry of Bulbar Origin, 62
- Tic: 584; Facial, 56; Hysterical, 144; Multiform, 220; Facial, cured by Suggestion, 220; of Childhood, Treatment of, 382; Convulsive, 444
- Tobacco, Ocular Symptoms due to, 837
- Tongue: Hemiatrophy of and Facial Paralysis, 289; Diphtheritic Paralysis of, 217
- Torticollis: Hysterical, 58
- Transplantation of Nerves; 386; 745
- Trapezius, Spasm of Right, 56
- Trauma: Dementia Paralytica after, 229; and General Paralysis, 307; 813; Consequences of Injury to Peripheral Nerves, 47; Isolated Paralysis due to, 499; and Pupil (3), 351
- Tremor: Intention, in Children, 62; in Frontal Tumours, 809
- Trypanosomiasis, 112
- Tumour: *Brain*: Mental Symptoms of Cerebral, 509; Anæsthesia, Attacks of Hemialgesia and Early Muscular Atrophy in, 835; Meningeal, 134; Pituitary, 429; 835; Psammoma of Dura Mater, 142; Cerebellar, 218; 510; Extracerebellar, 508; Localisation of Frontal, 809; Embryoma of Frontal Lobe, 338; Corpus Callosum, 694; Temporosphenoidal Lobe, 833; Results of Operation for Removal of, 782; Palliative Operation in, 786
- Tumour: *Spinal Cord*: Extra-Medullary New Growths, 359; Descending Degenerations of Posterior Columns after Compression of Dorsal Roots by, 438; Operation for Tumour of Spinal Meninges, 644
- Typhoid Fever, *vide* Enteric
- VASCULAR Lesions, Hemiplegia in, 365

- |  |  |
|--|--|
| <b>Venoms</b> : Action of, on Nervous System, 498<br><b>Vertigo</b> : in Epilepsy, 694<br><b>Vision</b> : Cortical Visual Area, 372 ; Projection of Retina on Cortex of Occipital Lobe, 372 ; Hyperæsthesia of Visual Periphery, 580 | <b>WALLERIAN Law</b> , 210<br>Weeping, Spasmodic, 371<br><b>Weigert's Neuroglia Stain</b> , 738<br><b>Westphal's Pseudo-sclerosis</b> , 831<br><b>Whooping-Cough</b> : followed by Hemiplegia, 54<br><b>Wrisberg, Nerve of</b> , 473 |
|--|--|

## INDEX OF AUTHORS.

- ALBRECHT. Manic-Depressive Insanity and Arterio-sclerosis, 707
- Alquier. Nervous Troubles in Pott's Disease, 359
- Alter. Hydrotherapy in Mental Disorders, 777
- Alzheimer. Degeneration of Nerve Tissue, 637
- Amblard. Pneumococcal Cerebro-spinal Meningitis and Diabetes, 752
- Anderson. Paralysis of Involuntary Muscle, 39, 279
- Anton. Symptoms of Frontal Disease, 646
- Archambault. Inferior Longitudinal Bundle and Central Optic Bundle, 31
- Arndt. Insight in Mental Diseases, 379
- Aschaffenburg. Variations of Mood in the Epileptic, 523
- Auerbach. Localisation of Musical Talent in Brain and on Skull, 536
- Avdskoff. Paralysis of External Branch of Spinal Accessory in Tabes, 439.
- Axenfeld. Traumatic Reflex Immobility of Pupil, 452
- BABES and Marinesco. Atlas of Pathological Histology of Nervous System (Review), 867
- Babinski. Neuritis, possibly due to Working with Artificial Manures, 50; Latent Forms of Affections of Pyramidal System, 54; Spasm of Right Trapezius and Facial Tic, 56; Peripheral Facial Hemispasm, 57; Thermo-Asymmetry of Bulbar Origin, 62; Paralysis by Compression of Pyramidal Tract, 693; Cerebellar Asynergy and Inertia, 703
- Babinski and Tofesco. "Blue Disease," Retinal Cyanosis, Hemiplegia following on Whooping-Cough, 54
- Bach. Decussation of Trochlear Nerve, 203; Motor Nuclei after Lesions of Peripheral Nerves, 288
- Bainbridge and Dale. Contractile Mechanism of Gall-bladder and its Extrinsic Nervous Control, 37
- Baird, Harvey. Pathology of Epileptic Idiocy, 689
- Balbeze. Kernig's Sign in Herpes Zoster, 300
- Bálint and Benedict. Diseases of Conus Terminalis and Cauda Equina, 292
- Barnes, Stanley, and E. Farquhar Buzzard. Chronic Progressive Double Hemiplegia, 183
- Barrett. Spinal Cord Degeneration in Acromegaly, with Tumour of Pituitary Region, 429; Mental Diseases associated with Cerebral Arterio-Sclerosis, 458; Disseminated Syphilitic Encephalitis, 644
- Batten, Fred. E. Cerebral Symptoms due to Encephalitis; Relation of Disease to Acute Anterior Poliomyelitis 140; Ataxia in Childhood, 297
- Baumann. Hysterical Twilight-State, 760
- Bechterew, W. v. What is Hypnosis? 445; Peculiar Reflex in Affection of Central Motor Neurons, 448
- Beebe. Serum for Treatment of Exophthalmic Goitre, 384
- Bellei. Mental Fatigue in School Children, 285
- Benedict and Bálint. Diseases of Conus Terminalis and Cauda Equina, 292
- Berliner. Histology and Developmental History of Cerebellum, 32; Cerebellar Tumour with Mental Symptoms, 218
- Bernhardt. Pathology of Exophthalmic Goitre, 647
- Bernstein, Julius, and Purves Stewart. Partial Doubling of Spinal Cord, 729
- Bertholet. Conducting Paths for Painful and Thermal Impressions in Cord, 424
- Besta, Carlo. Degeneration and Regeneration of Peripheral Nerve Fibres, 558
- Bianchi. Cerebral Cortex of Dolphin, 34
- Binswanger. Treatment of Mental Diseases, 63
- Bleuler. Affectivity, Suggestibility, and Paranoia (Review), 393
- Blumer, G. A. Coming of Psychasthenia, 518
- Boinet. Diver's Palsy, 755
- Bonfigli. Aphasia, 651
- Bonhoeffer. Jacksonian Epilepsy in Topical Cerebral Diagnosis, 647
- Borchardt and Oppenheim. Operation for Tumour of Spinal Meninges, 644
- Boschi and Graziani. Pott's Paraplegia, 752

- Bouchand. Infantile Cerebral Hemiplegia, 506
- Bowlby, Anthony. Surgical Complications of *Tabes Dorsalis*, 501
- Bradley, O. Charnock. Hind-Brain of Pig, 126
- Braillon. Pupil Reflexes in Mitral Valve Lesions, 581
- Bramwell, Byrom. Intracranial Aneurisms, 507; Extra-Cerebellar Tumours, 508.
- Bramwell, Edwin. Recognition of Segmental Levels in Cervical and Lumbar Enlargements of Cord from Appearance of Transverse Section, 344; Juvenile General Paralysis, 313
- Bramwell, Edwin, and Arthur H. H. Sinclair. Ophthalmoplegia interna unilaterale, 841
- Bratz and Leubuscher. Epilepsy with Unilateral Manifestations, 696
- Briessaud, Sicard and Tanon. Treatment of Contractures, Spasms and Tremors by Local Injection of Alcohol into Nerve Trunks, 710
- Brower. Treatment of Acute Insanity in a General Hospital, 709
- Brown, H. Egerton. *Cysticercus Cellulose* in the Insane, 373
- Browning, William. Spinal Hemorrhage, 137; Treatment of Cerebro-Spinal Meningitis, 234
- Bruce, Alexander. Distribution of Cells in Intermedio-lateral Tract, 349
- Bruce, Alexander, Stuart M'Donald, and J. H. Harvey Pirie. Partial Doubling of Spinal Cord, 6
- Bruce, Lewis C. Serum Reaction in Persons suffering from Infective Conditions, 562
- Bruel. Tics and Chorea of Childhood, 382
- Bryant. Psychological Importance of Ear Disease, 776
- Buck, J. de, and Deroubaix. Histopathology of Certain Forms of Psychosis allied to *Dementia Præcox*, 287
- Bullard. Operation in Head Injuries, 463
- Bumke. Immobility of Pupil in Hysterical Attacks, 453; "Zwangsvorgänge," 460
- Burns, R. L., Stewart, Guthrie, and Pike. Resuscitation of Central Nervous System of Mammals, 741
- Buzzard, E. Farquhar. Myasthenia Gravis, 295
- Buzzard, E. Farquhar, and J. Cuning. Post-Traumatic Hemorrhage from Superior Longitudinal Sinus without Fracture of Skull, 443
- Buzzard, E. Farquhar, and Stanley Barnes. Chronic Progressive Double Hemiplegia, 182
- Buzzard, Thomas. Diagnosis of Tumour or other Lesion of Uncinate Region of Temporo-sphenoidal Lobe, 833
- CAJAL, S. R. Cell Types in Sensory Ganglia, 124; Nerve Regeneration, 134
- Campbell, Alfred W. Localisation of Cerebral Function (Review), 308
- Campbell. Cerebral Sclerosis, 427
- Campbell, C. Macfie. Muscular Dystrophy affecting Hands and Feet, 192
- Camus and Dupré. Pott's Paraplegia, 218
- Cans and Rodut. Diagnosis of Cerebral Disturbances of Toxic Origin and General Paralysis by Ocular Symptoms, 837
- Cantonnet. Paralysis of Associated Movements of Eyes, 454
- Cardenal, de, and Verger. *Tabes*, 692
- Catolà. Disseminated Sclerosis, Cerebellar Atrophy, Pseudo-Systemic Sclerosis of Cord, 139; Pathology of Parkinson's Disease, 689
- Ceni, Carlo. Anatomical Localisation of Delirium produced by Toxines of Pellagra, 45
- Chaillons and Pagniez. Bilateral Ophthalmoplegia Externa, 302
- Chartier and Lejonne. Ascending Neuritis and Chronic Rheumatism, 828
- Chotzen. Mixed Conditions in Epilepsy and Alcoholism, 589; Atypical Alcoholic Psychosis, 705
- Church. Neuritic Type of Progressive Muscular Atrophy, 691
- Ciacco, Carmello. Reproduction of Nerve Cells, 820
- Clarke, J. Michell. Myasthenia Gravis, 146
- Cohn, Toby. Die palpablen Gebilde des normalen menschlichen Körpers (Review), 465
- Cole, Sidney J. Aphasia and Mental Disease, 304
- Conzen. Arsenical Neuritis, 216
- Coriat. Alcoholic Neuritis, 564
- Cramer and Long. Late *Tabes*, 289
- Crocq. *Dementia Præcox*, 848
- Cruchet. Peripheral Post-Paralytic Hemispasm, 56; Convulsive Tic, 444
- Cruchet and Pitres. Hysterical Tic, 144
- Cumming, J., and E. Farquhar Buzzard. Hemorrhage from Superior Longitudinal Sinus without Fracture of Skull, 443
- Curtis, B. Farquhar. Surgical Treatment of Exophthalmic Goitre, 462
- Cushing, Harvey. Surgical Intervention

- for Intra-cranial Hæmorrhages of New-born, 66; Sexual Infantilism with Optic Atrophy in Tumour of Hypophysis Cerebri, 835
- D'ABUNDO, G. Experimental Cerebral Atrophies and Accompanying Cranial Atrophies, 742
- Daireaux. Paralysis of Pneumonia in Adults, 767
- Dale and Bainbridge. Contractile Mechanism of Gall-Bladder and its Extrinsic Nervous Control, 37
- Dannemann. Psychic and Motor Disturbances caused by Alcohol, 591
- Dannenberg. Porencephalic Form of Infantile Paralysis, 571
- Debove. Tabes and Aortic Aneurysm, 565; Hysterical Dysarthria, 654
- Debray. Sensory Aphasia, with Right Homonymous Lateral Hemianopia, 377
- Déjerine. Intermittent Claudication of Spinal Cord, 584; Sensory Aphasia, its Localisation and Pathological Physiology, 650; "Tactile Aphasia," 708
- Déjerine, M. et Mme. Cell Columns of Anterior Cornua of Spinal Cord, 737
- Déjerine and Roussy. Thalamic Syndrome, 648
- Delacroix and Solager. Retro-antegrade Amnesia, etc., in Hysteria, 223
- Deny, G. Manic-Depressive Insanity, 849
- Dercum. Heboid Paranoid Group of Dementia Præcox, 708
- Deroubaix. Spasmodic Laughter and Weeping, 371
- Deroubaix and de Buck. Histopathology of Certain Forms of Psychosis allied to Dementia Præcox, 287
- Devaux. Osmotic Theory of Sleep, 423
- Donaggio. Pyridine Methods for Rapid Differentiation of Reticulum of Nerve Elements, 684; Effects of Combined Fasting and Cold on Nerve Centres, 687
- Donath. Choline in Cerebro-spinal Fluid, 285
- Dontas and Mavrakis. Respiratory Centre in Cerebral Cortex of Dog, 35
- Dorleans. Co-existence of Tertiary Syphilitic Lesions with Tabes and General Paralysis, 748
- Drew. Common Form of Insanity, 457
- Dreyfus. Traumatic Immobility of Pupil (2), 451
- Dupré and Camus. Pott's Paraplegia, 218
- EDGEWORTH. Transitory Hemiplegia in Elderly Persons, 838
- Egger and Raymond. Tactile Aphasia, 586
- Ellerman. Rhizopods in Acute Anterior Poliomyelitis, 353
- Elliott. Degenerative Section of Nerves to Cat's Bladder, 275
- Erb. Intermittent Claudication, 153, 698
- Esposito. Psammomata of Dura Mater, 142
- Étienne. Intense Cold in Pathogeny of Acropathies, 155
- Eulenburg. Antithyreoidin Treatment of Exophthalmic Goitre, 65
- Evensen, Hans. Pathology of General Paralysis, 537, 616
- FARRAR, Clarence B. Devolutional Psychoses, 665
- Faure, Maurice. Laryngeal Crises of Tabes, 692
- Federn. Blood-Pressure and Neurasthenia, 58
- Feix. Method of Investigation of Knee- and Ankle-Jerks, 844
- Fels. Symptom-Complex of Primary Incoherence with Excitement, 775
- Féré, Ch. Rest in Ergographic Work, Mechanical Value of Mental Representation of Movement, etc., 283; False Reminiscence, 847
- Ferguson, Alex. Hugh. Ischemic Muscular Atrophy, Contractures, and Paralysis, 514
- Ferrier, David. Tabes Dorsalis, 433
- Fichera. Experimental Destruction of Hypophysis, 207
- Fischer, Oskar. Isolated Palsy for Lateral Movement of Internal Rectus Muscle, 375; Macropsia and its Relation to Micrographia, and Peculiar Alteration in Light Perception, 449; Heart Neuroses and Basedow's Disease, 697
- Fischler. Isolated Traumatic Paralysis, 499; Traumatic Conus Lesions, 567
- Fitzgerald. Lumbo-Sacral-Coccygeal Cord of Macaque Monkey, 683
- Flashman, J. F. Motor Areas in Cerebral Cortex of *Dasyurus Viverrinus*, 635; Brain of Microcephalic Idiot, showing lack of Corpus Callosum, 639
- Fleig. Muscular-Tonic Action of Formic Acid, 232
- Flexner, Simon. Experimental Cerebro-spinal Meningitis and its Serum Treatment, 826
- Forli. Rheumatic Polyneuritis of Cranial Nerves, 690
- Forli and Guidi. Pharyngeal Reflex, 154

- Français and Raymond. Syringomyelia with Sensory Disturbances of Radicular Distribution, 442
- Frankl-Hochwart. Prognosis of Adult Tetany, 698
- Franz, S. I. Time of Some Mental Processes in Retardation and Excitement of Insanity, 303
- Frazier and Spiller. Cerebral Decompression, 786
- Frazier, Spiller, and Van Kaathoven. Treatment of Nerve Palsies and Athetosis by Nerve Transplantation, 386
- Freud. Hysteria Analysis, 522
- Frey, Ernest. Post-Hemiplegic Movements, 368
- Frey, Max von. Distribution of Afferent Nerves in Skin, 738
- Friedmann. Non-Epileptic Affections of Consciousness or Short Narcoleptic Attacks, 575
- Fuhrmann. Acute Juvenile Deterioration, 225
- GALEZOWSKI. Bi-temporal Hemianopia, 699 ; 777
- Galezowski, Raymond, and Lejonne. Blindness of Cortical Origin from Double Hemianopia, 699
- Gardner, Eric. Family in which Signs of Friedreich's Ataxy appeared Discretely, 441
- Gaupp. Depressions of Advanced Life, 159 ; Crimes committed during Intoxication, 592
- Gaussel. Acromegaly, with Lesion of Hypophysis and Sella Turcica, 759
- Gehuchten, A. van. Faisceau en Crochet, 203 ; The Wallerian Law, 210
- Geiravold. Bacteriology of Acute Anterior Poliomyelitis, 132
- Geist. Lobus Cerebelli Medianus, 737
- Gemelli. Structure of Motor Nerve-endings in Reptiles, 202 ; Structure of the Nerve Cell, 556
- Gieseller. General Paralysis and Trauma, 307
- Goldscheider. On Organic Changes in "Assoziationsbildung," 278 ; Sensory Ataxia, 446
- Goldscheider and Leyden. Diseases of Cord and Medulla Oblongata (Review), 162
- Gordon, Alfred. Pathogenesis of Exophthalmic Goitre, 59
- Gottgetreu. Psychoses of Children, 461
- Gowers, Sir W. R. Influence of Facial Hemiatrophy on Facial and other Nerves, 1 ; Chorea and its Neuronic Aspect, 219 ; Tetanoid Chorea and its Association with Cirrhosis of the Liver, 249 ; Dystrophy of Tabes and Problem of Trophic Nerves, 437 ; Dendrites and Diseases, 636 ; Borderland of Epilepsy, 694, 696
- Grabower. Zur Recurrensfrage, 769
- Grasset. Intermittent Claudication of Nervous Centres, 585
- Graziani and Boechi. Pott's Paraplegia, 752
- Green, D. Crosby. The Larynx in Tabes, 290
- Groszmann. Position of the Atypical Child, 656
- Gudden. "Sleep Drunkenness," 229.
- Guerrini. Function of Degenerate Muscles, 214, 559
- Guidi and Forli. Pharyngeal Reflex, 154
- Guillain and Raymond. Syringobulbia, 368
- Guizzetti. Chromatic Pseudo-Corpuscles of Axis-Cylinder, 123
- Guthrie, C. C., Stewart, Burns, and Pike. Resuscitation of Central Nervous System of Mammals, 741
- HALLIBURTON and Mott. Suprarenal Capsules in Nervous Diseases, 352
- Hamburger. Diphtheritic Paralysis of Left Hypoglossus, 217
- Hare, Francis. Food Factor in Paroxysmal Neuroses, 236
- Haskovec. Ocular Crises in Tabes, 642
- Head, Henry. Afferent Nervous System from New Aspect, 47
- Head, Henry, and James Sherren. Consequences of Injury to Peripheral Nerves, 47
- Hecht. Myoclonus Multiplex, 143 ; Dementia Præcox, 302
- Heilbronner. Agrammatismus and Disturbance of Internal Language, 652 ; Progressive Paralysis of Insane, 847
- Heitz and Roux. Effect of Experimental Section of Posterior Roots upon Peripheral Neurons, 743
- Heller. Multiple Sclerosis and the Psychogenic Neuroses, 831
- Hirt, Eduard. The Temperaments (Review), 240
- Hoch, August. Delirium produced by Drugs, 83
- Holmes, Gordon, F. J. Poynton, and J. H. Parsons. Amaurotic Family Idiocy, 568
- Hoppe, Fritz. Weigert's Neuroglia Stain, 738
- Hoppe, Herm. H. Hysterical Stigmata caused by Organic Brain Lesions, 297 ; Treatment of Crimes committed by

- Alcoholic, 593; Walk through a Modern Asylum (Review), 870
- Horsley, Sir Victor. The Tænia Pontis, 413; Technique of Operations on Central Nervous System, 778
- Houzel. Bleeding in Epilepsy, 383
- Howard. Relation of Lesions of Gasserian and Posterior Root Ganglia to Herpes occurring in Pneumonia and Cerebro-spinal Meningitis, 139; Tetany, 762
- Hübner. Psychical and Sensory Reaction of Pupils, 149; Tabes, General Paralysis, and Syphilis, 356
- Hudovernig. Hallucinations of Peripheral Origin, 776
- Huet and Lejonne. Facial Paralysis and Hemiatrophy of Tongue, 289
- Humphrey, Lawrence. Parathyroid Glands in Graves' Disease, 46
- Hunter, W. K., and George Lamb. Action of Snake Venoms on Nervous System, 498
- Hunter, Walter K., and Charles Workman. Cord and Medulla in Acute Ascending Paralysis, 106
- ИОТЕУКО. Laws of Ergography, 40, 128, 280, 282; Sense of Pain, 149; Facial Tic cured by Suggestion, 220; Mathematical Analysis of Fatigue Curves in Diagnosis of Nervous Diseases, 224
- JACKSON, Edward. Developmental Alexia, 518
- Jaeger. Familial Cretinism, 224
- Janet, P. Pathogenesis of some Impulses, 459
- Jelliffe. Aphasia, Hemiparesis, and Hemianæsthesia in Migraine, 223
- Jochmann. Recurrent Third Nerve Paralysis in Enteric Fever, 454
- Johnston, J. B. Radix Mesencephalica Trigemina and Ganglion Isthmi, 415; Cranial Nerve Components of Petro-myzon, 418; Cranial and Spinal Ganglia and Viscero-motor Roots in Amphioxus, 419
- Jones, A. Ernest. Hemiplegia in Vascular Lesions, 365
- Jordan-Lloyd. Facial Neuralgia treated by Excision of Gasserian Ganglion, 793
- Juliusburger. Want of Insight in Alcoholics, 589; Treatment of Crimes committed by Alcoholics, 593
- Jung, C. G. Disclosure of Fact by Psychological Methods, 305; Experimental Observations on Memory, 305; Diagnostic Association Studies, 520
- KAATHOVEN, Van, Spiller, and Frazier. Treatment of Nerve Palsies and Athetosis by Nerve Transplantation, 386
- Kaes, Theodor. Width of Cortex in Estimation of Brain Development and Intelligence, 33
- Kast. Head's Zones in Disorders of Digestive Organs, 701
- Kiroff. Babinski's Sign in Scarlet Fever, 151
- Klippel and Lhermitte. The Dementias, 382
- Klippel and Villaret. The Asthenias and Myopathic Atrophies, 295
- Klipstein. Hebeephrenic Form of Dementia Præcox, 707
- Knapp, Philip Coombs. Mental Symptoms of Cerebral Tumour, 509; Results of Removal of Cerebral Tumours, 782
- Kollarita. Hysterical Torticollis, 58
- Kovalesky. Epilepsy and Migraine, 839
- Kramer, Franz. Cortical Tactile Paralysis, 299
- Krause. Extirpation of Gasserian Ganglion, 795
- Kreuzfuchs. Traumatic Immobility of Pupil, 451
- Krönig. Device for Obtaining Knee-Jerk, 844
- Kümmel. Operative Treatment of Purulent Meningitis, 791
- LACHE. Post-mortem Changes in Neurofibrils, 352
- Lamb, George, and W. K. Hunter. Action of Snake Venoms on Nervous System, 498
- Lambrior and Puscariu. Amyotrophic Lateral Sclerosis, 750
- Lamy. Synergic Paradoxical Contractions following Peripheral Facial Palsy, 57
- Langley, J. N. Reaction of Cells and Nerve-endings to Certain Poisons, 276
- Langwill, H. G. Transitory Hemiplegia, 505
- Lapinsky. Forms of Tabes Seldom Described, 358
- La Place. Treatment of Idiopathic Epilepsy by Appendicostomy for Colonic Irrigation, 579
- Laruelle. Position of Head in Cerebellar Disease, etc., 455
- Lasarew. Juvenile Tabes, 136; Schäfer's Antagonistic Reflex, 448
- Lejonne and Chartier. Ascending Neuritis and Chronic Rheumatism, 828
- Lejonne and Huet. Facial Paralysis and Hemiatrophy of Tongue, 289



- Lejonne and Lhermitte. Lacunar and Myelopathic Paraplegia in the Old, 58; Paraplegias from Retraction in Old People, 566
- Lejonne and Raymond. Pseudo-Bulbar Palsy in a Child, 584
- Lejonne, Raymond, and Galezowski. Blindness of Cortical Origin from Double Hemianopia, 699
- Léri, André. The Senile Brain, 753
- Léri, André, and Pierre Marie. Spondylitis Deformans, 361
- Leroy. Responsibility of Hysterics, 760
- Letessier and Sabrazès. Method of Staining Neuroglia, 205
- Leubuscher and Bratz. Epilepsy with Unilateral Manifestations, 696
- Leupoldt, Curt von. Catatonia, 226; Investigation of Traumatic Neuroses by Psycho-Physical Methods, 587
- Lewandowsky. Disturbances of Movement in Infantile Cerebral Hemiplegia; Double Athetosis, 146; Apraxia in General Paralysis, 306; Projection of Pain Sensation from Lower to Upper Extremity, 702
- Leyden and Goldscheider. Diseases of Cord and Medulla Oblongata (Review), 162
- Lhermitte and Klippel. The Dementias, 382
- Lhermitte and Lejonne. Lacunar and Myelopathic Paraplegia in the Old, 58; Paraplegias from Retraction in Old People, 566
- Liepmann. Minor Aids in Examination of Brain Disease, 52
- Lilienstein. Exhaustion from Excess of Function, 424
- Lograsso and Onuf. Blood of Epileptics, 286
- Lomer. Relation of Psychoses of Involution Period to Juvenile Dementia, 456
- Long and Cramer. Late Tabes, 289
- Long and Revilliod. Polyneuritis as Sequela of German Measles, 356
- Lorenz. Transplantation of Tendons, 287
- Lucas, Keith. Gradation of Activity in Skeletal Muscle-Fibre, 127
- Lugaro. Non-Occurrence of Autogenous Regeneration of Nerve Fibres, 637; Neurotropism and Transplantation of Nerves, 745
- MAINGER. "Hysteria" of Animals, 513
- Marbe and Noica. Cutaneous and Spinal Reflexes in Spastic Paraplegia, 700
- Margaria, Guiseppe. Etiology and Duration of General Paralysis, 645
- Marie. Supposed Immunity of Syphilitic Arabs regarding General Paralysis, 513; Aphasia: Third Left Frontal Convolution does not play Special Rôle in Function of Speech, 649; Subcortical Aphasia, 845
- Marie, Pierre, and André Léri. Spondylitis Deformans, 361
- Marie and Montier. Pontine Hæmorrhage, 754
- Marie and Pelletier. Perforating Ulcer in General Paralysis, 369
- Marinesco. Autogenic Regeneration, 209; Disturbances of Baræsthesia, or Sense of Pressure, 446; Reticulated Structure of Axis-Cylinder, 737
- Marinesco and Babes. Atlas of Pathological Histology of Nervous System (Review), 867
- Marinesco and Minea. Regeneration in Peripheral Nerves, 425
- Marshall, D. G. Trypanosomiasis or Sleeping Sickness, 113
- Mattirolo. Rare Form of Pseudo-æsthesia, 768
- Mavrikis and Dontas. Respiratory Centre in Cerebral Cortex of Dog, 35
- M'Bride, P. Deafness due to Hysteria, etc., 516
- M'Donald, Stuart, Alexander Bruce, and J. H. Harvey Pirie. Partial Doubling of Spinal Cord, 6
- Mackenzie, James. Visceral Pain, 700
- Mackey. Osteitis Deformans with Huntingdon's Chorea, 840
- Mackintosh, Ashley. Frequency of Certain Signs and Symptoms in Disseminated Sclerosis, 601
- Mendel, Kurt. Dorsal Foot Reflex, 447
- Merzbacher. Lumbar Puncture in Psychiatry and Neurology, 655; Relation of Syphilis to Lymphocytosis of the Cerebro-spinal Fluid and to the Question of "Meningeal Irritation," 749
- Mills, C. K. Crural Monoplegia, 296; Jacksonian Epilepsy in Focal Diagnosis, 511
- Mills and Weisenburg. Word-Blindness, Treatment of Visual Aphasia, 152; Localisation of Higher Psychic Functions, 420; Cortical Representation of Cutaneous and Muscular Sensibility, 819
- Milne, C. J. Robertson. Epidemic Cerebro-Spinal Meningitis in India, 361
- Minciotti. Philipovitch's Sign in Enteric Fever, 378
- Minea and Marinesco. Regeneration in Peripheral Nerves, 425
- Minor. Pathology of Epiconus Medullaris, 567

- Mirallée. Peripheral Facial Palsy, 690  
 Möbius. Robert Schumann's Disease (Review), 526  
 Moeli. Discharge of Patients from Asylums in Prussia, 851  
 Moleen. Chronic Anterior Poliomyelitis, 137  
 Montier and Marie. Pontine Hæmorrhage, 754  
 Montyel, Marandon de. Pupil Accommodation Reflex in General Paralysis, 307  
 Moon. Convulsions in Children and their Relation to Epilepsy, 839  
 Morawitz. Multiple Sclerosis in Guise of Transverse Myelitis, 51  
 Mörohen. Pal's Teaching as to Vascular Crises of Tabetics, 830  
 Moschcowitz. Surgical Treatment of Trigeminal Neuralgia, 857  
 Mott and Halliburton. Suprarenal Capsules in Nervous Diseases, 352  
 Mourre. Nerve Cells, studied by Nissl's Method, 123  
 Müller. Functions of Bladder in Cerebral Hemiplegia, 152; Extirpation of Lower Half of Spinal Cord, 562  
 Münzer. Puerperal Psychoses, 588
- NÄCKE. Late Epilepsy in Chronic Psychoses, 381  
 Nadjede and Parhon. Hemieranosia, 61; Localisation in Nuclei of Cranial and Spinal Nerves, 416  
 Nageotte, J. Nervous Intermedius of Wrisberg and Bulbo-Pontine Nucleus, 473; Collateral Regeneration by Nerve Fibrils with Club-like Terminals, 825  
 Neisser. Confabulation, 228  
 Neumann. Hæmatemesis in Organic Nervous Diseases, 154  
 Nikolaides. Central Respiratory Innervation, 36  
 Nissl, Franz. Histology of Cerebral Cortex and Pathological Anatomy of Mental Diseases (Review), 387  
 Noica. Primary Progressive Myopathy in Two Brothers, 563  
 Noica and Marbe. Cutaneous and Tendon Reflexes in Spastic Paraplegia, 700.  
 Nonne. Anatomical Basis of Syphilitic Spinal Paralysis, 211  
 Norman, Conolly. Multiple Lipomata in General Paralysis, 369
- OBICI. Method of Inscribing Tracings on Revolving Cylinder, 208  
 Onuf and Lograsso. Blood of Epileptics, 286  
 Oppenheim. Extra-Medullary Spinal New-Growths, 359  
 Oppenheim. Psychotherapeutic Letters (Review), 657  
 Oppenheim and Borchardt. Operation for Tumour of Spinal Meninges, 644  
 Orchansky. Tabes Dorsalis and Knee Angle Phenomenon, 501  
 Orr, David. Descending Degenerations of Posterior Column in Transverse Myelitis and Compression of Dorsal Posterior Roots by Tumours, 488  
 Orr, David, and R. G. Rows. Lesions of Spinal Cord, Result of Absorption from Localised Septic Foci, 25  
 Ortali. Abdominal Reflex in Enteric Fever, 222  
 Osler, William. Convulsions in Typhoid, 143  
 Ostermann. Meningococcal Pharyngitis in Epidemic Cerebro-spinal Meningitis, 354  
 Owen, John, and W. B. Warrington. Pathology of Myelitis Acutissima Hæmorrhagica Disseminata, 401; Hæmorrhage into Brain and Cord from Obliterative Arterial Disease, 407
- PACKARD. Feeling of Unreality, 519  
 Pagano. Functions of Caudate Nucleus, 817  
 Pagniez and Chaillons. Bilateral Ophthalmoplegia Externa, 302  
 Pailhas. Baths and Hydrotherapy in Treatment of Mental Disease, 64  
 Paine and Poynton. Nervous Manifestations of Acute Rheumatism, 59  
 Panichi. Pathogenesis of Paralysis induced by Infection with Pneumococci, 44  
 Parhon and Nadjede. Hemieranosia, 61; Localisation in Nuclei of Cranial and Spinal Nerves, 416  
 Parsons, Poynton, and Gordon Holmes. Amaurotic Family Idiocy, 568  
 Paul and Walton. Cerebral Element in Reflexes; its Relation to Spinal Element, 843  
 Pawlow. Psychical Faculties or Processes in Higher Animals, 822  
 Pelletier and Marie. Perforating Ulcer in General Paralysis, 369  
 Pelizzi. Forms of Idiocy due to Meningitis, 156; Heterotopia of Cerebral Cortical Substance, 428  
 Pers, Alfred. Surgical Treatment of Sciatica, 465  
 Pfeiffer. Circumscribed Autopsychosis on Basis of a Morbidly Dominant Idea, 461  
 Phelps, Charles. Function of Left Prefrontal Lobe, 362  
 Pick. Retrogression of Hemianopic

- Disorders after Paralytic Attacks, 148 ; Psychology of Confabulation, 227 ; Mixed Apraxia, 380 ; Hyperaesthesia of Visual Periphery, 580
- Pike, F. H., Stewart, Guthrie, and Burns. Resuscitation of Central Nervous System of Mammals, 741
- Pitres and Cruchet. Hysterical Tic, 144
- Pirie, J. H. Harvey, Alexander Bruce, and Stuart M'Donald. Partial Doubling of Spinal Cord, 6
- Potter, N. B. Atrophy of Glands at Base of Tongue as sign of Syphilis, 588
- Poynton and Paine. Nervous Manifestations of Acute Rheumatism, 59
- Poynton, Parsons, and Gordon Holmes. Amaurotic Family Idiocy, 568
- Primangeli. Transverse Myelitis as a Sequela of Measles, 751
- Prince, Morton. Multiform Tic, 220 ; Psychology of Sudden Religious Conversion, 457 ; Head Injuries, 468 ; Journal of Abnormal Psychology (Review), 527 ; Limited Area of Anæsthesia, etc., in Brain Tumour, 835 ; Hysteria from Point of View of Dissociated Personality, 840
- Pringle, J. Hogarth. Hæmorrhage from Middle Meningeal Artery, 364
- Puscarin and Lambrior. Amyotrophic Lateral Sclerosis, 750
- Putnam, J. J. Hysteria in Massachusetts General Hospital, 462
- Putnam and Waterman. Cerebellar Tumours, 510
- RAMSAY, A. Maitland, and W. M. Sutherland. Spindle-Shaped Enlargement of Blind-Spot with Congestion of Optic Disc, 301
- Ranson, S. Walter. Retrograde Degeneration in Spinal Nerves, 686, 747
- Raymond. Tumour of Corpus Callosum, 694
- Raymond and Egger. Tactile Aphasia, 586
- Raymond and François. Syringomyelia with Sensory Disturbances of Radicular Distribution, 442
- Raymond and Guillain. Syringobulbia, 368
- Raymond and Lejonne. Pseudo-Bulbar Palsy in a Child, 584
- Raymond, Lejonne, and Galezowski. Blindness of Cortical Origin, from Double Hemianopia, 699
- Ræcke. Mental Disorders in Multiple Sclerosis, 642
- Rebizzi. Toxic Cause in Mental Disease, 640
- Redlich, Emil. Etiology of Epilepsy, 574
- Régis. Mental Confusion, 379 ; Traumatic Neurasthenia in Arterio-Sclerosis, 761
- Reichardt. Delirium Tremens, 231 ; Estimation of Skull Capacity on Cadaver, 420
- Reinhold. Dementia Paralytica after Trauma, 229
- Rendu, A., Jules Voisin, and Roger Voisin. Idiocy and Cerebellar Lesion, 523
- Revilliod and Long. Polyneuritis as Sequela of German Measles, 356
- Reynolds, Ernest S. Paramyoclonus Epilepticus, 19
- Riegner. Physiology of Jaw-Movements, 558
- Riklin. Symptoms and Associations of Hysteria, 293
- Riva. Neuro-fibrillary Reticulum in Experimental Inanition, 687
- Robertson, W. Ford. Pathology of General Paralysis of Insane, 73, 169, 258
- Rodrigues. Polyneuritic Psychosis and Beri-beri, 848
- Rodut and Cans. Diagnosis of Cerebral Disturbances of Toxic Origin and General Paralysis by Ocular Symptoms, 837
- Roemheld. Korsakow's Symptom-Complex in Brain Syphilis, 704
- Rogers, John. Serum Treatment of Exophthalmic Goitre, 384
- Rolleston, J. D. Abdominal Reflex in Enteric Fever, 582 ; Precocious Paralysis of Palate in Diphtheria, 606
- Rosanoff. Diet in Epilepsy, 235
- Rosenheim. Preparation of Cholesterin from Brain, 351
- Ross. Peculiar Attitudes in Epilepsy during Sleep, 839
- Rossi. Structure of Nerve Cell in Vertebrates, 416
- Rossi and Roussy. Pathology of Amyotrophic Lateral Sclerosis, etc., 638
- Roth. White Rami Fibres and Spinal Accessory Nerve, 33
- Rothmann, Max. Sensory Conduction in Spinal Cord, 205
- Roubinovitch. Tic, 584
- Roussy. Meningeal Tumours, 134
- Roussy and Déjerine. Thalamic Syndrome, 648
- Roussy and Rossi. Pathology of Amyotrophic Lateral Sclerosis, etc., 638
- Roux and Heitz. Effect of Experimental Section of Posterior Roots upon Peripheral Neurona, 748

- Rowe, R. G. Embryoma in Frontal Lobe, 338
- Rowe, R. G., and David Orr. Lesions of Spinal Cord, Result of Absorption from Localised Septic Foci, 25
- Ruppel. Diplococcus Intracellularis Meningitidis and its Relations to Gonococci, 740
- Russell, A. E. Cessation of Pulse during Onset of Epileptic Fits, 579
- Russell, William. Cerebral Manifestations of Hypertonus in Sclerosed Arteries, 362
- SABRAZÈS and Letessier. Method of Staining Neuroglia, 205
- Sachs. Head Injuries and Operative Treatment, 463
- Salgó. Forensic Import of Sexual Perversity, 850
- Salle. Extension of Ascending Myelitis, 741
- Salomonson, W. Toxic Polyneuritis in a Phthisical Patient, 500
- Sandberg. Sensory Disturbances in Cerebral Hemiplegia, 367
- Santesson. Local Action of Cocaine and Stovaine on Peripheral Nerves, 828
- Sauerbeck. Malformation of Brain in Hatteria Punctata, 214
- Saxl. Extensor Phenomenon, 152
- Scarpini. Nerve Cells studied by Donaggio's Method, 557; Endocellular Fibrillary Reticulum and Long Fibrils in Cells of Spinal Cord, 557
- Schaffer. Fibrillar Structure in Progressive Paralysis, 212; Fibrillo-Reticular Substance in Swollen Nerve Cells, 739
- Schlesinger. Sensation in Acute Localised Ischæmia, 151; Palatal Paralysis, 221; Bilateral Circumscribed Facial Atrophy, 221
- Schloesser. Treatment of Neuralgias by Alcohol Injections, 236
- Schüller. Experimental Section of Pyramids in Dogs and Apes, 207
- Schultz, Paul. Gehirn und Seele (Review), 528
- Schultze, Kurt. Surgery of Basedow's Disease, 711
- Schutze. Landry's Paralysis after Typhoid, 291
- Scott, T. H. Metabolism and Action of Nerve Cells, 277
- Sherrin, James, and Henry Head. Consequences of Injury to Peripheral Nerves, 47
- Sherran, James. Distribution and Recovery of Peripheral Nerves, 430; Surgery of Peripheral Nerves, 860
- Sicard, Brissaud, and Tanon. Treatment of Contractures, Spasms, and Tremors by Local Injection of Alcohol into Nerve Trunks, 710
- Simpson. Westphal's Pseudo-Sclerosis, 831
- Sinclair, Arthur H. H., and Edwin Bramwell. Ophthalmoplegia interna unilaterale, 841
- Sinkler, Wharton, Friedreich's Ataxia, 217; Landry's Paralysis, 829
- Skłodowski. Treatment of Basedow's Disease with Röntgen Rays, 711
- Soca. "Crutch Paralysis," 564
- Solager and Delacroix. Retro-antegrade Amnesia, etc., in Hysteria, 223
- Sollier, Paul. Intermittent Claudication of Cord, 842
- Sommer. Psychiatric Observations on Case of Murder and Suicide, 231; Klinik für psychische und nervöse Krankheiten (Review), 238
- Southard. Neuroglia Framework of Cerebellum, 683
- Specht. Hystero-melancholia, 774
- Spielmeyer. Amaurotic Family Idiocy, 570
- Spiller, W. G. Separate Sensory Centres in Parietal Lobe for the Limbs, 296; Lesions of Left First Temporal Convolution in Relation to Sensory Aphasia, 329; Syringomyelia, 832
- Spiller and Frazier. Cerebral Decompression, 786
- Spiller, Frazier, and Van Kaathoven. Treatment of Nerve Palsies and Athetosis by Nerve Transplantation, 386
- Starr, Allan. Cerebellar Apoplexy, 506; Present Status of Brain Surgery, 852
- Sterling, W. Amaurotic Family Idiocy, 571
- Stewart, G. N., Guthrie, Burns, and Pike. Resuscitation of Central Nervous System of Mammals, 741
- Stewart, Purves. Clinical Significance of Cerebro-Spinal Fluid, 504; Diagnosis of Nervous Diseases (Review), 868
- Stewart, Purves, and Julius Bernstein. Partial Doubling of Spinal Cord, 729
- Stewart, T. Grainger. Localisation of Tumours of Frontal Region of Brain, 809
- Stiefler. Neural Form of Progressive Muscular Atrophy, 829
- Still, George F. Habit Spasm in Children, 145; Head-Nodding with Nystagmus in Infancy, 771
- Stoddart. Instinct, 686
- Sträussler. Cerebellum in General Paralysis, 561; Miliary Disseminated Form of Brain Syphilis and General Paralysis, 572

- Streeter, G. L. Cranial and Spinal Nerves in Occipital Region, 30; Spinal Cord of Ostrich, 34
- Strohmeyer. Amentia, 772
- Strümpell. Hysterical Fever, 443
- Sutherland, W. M., and A. Maitland Ramsay. Spindle-Shaped Enlargement of Blind-Spot with Congestion of Optic Disc, 301
- Symington, Johnson. Topographical Anatomy of Caput Gyri Hippocampi, 414
- TANON, Brissaud, and Sicard. Treatment of Contractures, Spasms, and Tremors by Local Injection of Alcohol into Nerve Trunks, 710
- Taylor, E. W. Multiple Sclerosis, 502
- Tebb, M. Christine. Cholesterin of Brain, 351
- Terson. Paralysis of Abducens in Otitis, 765
- Thoma. Slightly Abnormal Children, 380
- Thomas, André. Atrophy of Cells of Purkinje, 55; Ramon y Cajal's Method in Pathological Anatomy of the Axis-cylinder, 420
- Thompson, W. Gilman. Exophthalmic Goitre, 445
- Thomson, St Clair. Cerebral and Ophthalmic Complications in Sphenoidal Sinusitis, 757
- Thorington and Weisenburg. Syringomyelia with Double Optic Neuritis, 138
- Tiedemann. Poliomyelitis Anterior Acuta and Meningitis Cerebro-spinalis, 830
- Tödter. Paralysis of Movement Upwards and Downwards, 763
- Toufessoo and Babinski. "Blue Disease," Retinal Cyanosis, Hemiplegia following on Whooping-Cough, 54
- Trendelenburg. Movements of Birds after Section of Posterior Spinal Roots, 821
- Trevelyan, E. F. Infantile Paralysis, 440
- Trolard. Claustrium, 31
- Trömmner. "Stammering Gait," 770
- Tubby, A. H. Surgical Treatment of Paralysis, 384
- Turner, John. Pathology of Epilepsy, 354; Structure of Olfactory Bulb and Cornu Ammonis, as revealed by Pseudo-Vital Method, 417
- Turner, W. Aldren. Dietetic Treatment in Epilepsy, 383
- URBACH. Intention Tremor in Children, 62
- Urbautschitsch. Reflex Epilepsy, 338
- VASSALE. Puerperal Eclampsia and Parathyroid Insufficiency, 577
- Verger and de Cardenal. Tabes, 692]
- Vigouroux. Bed-Sores in General Paralysis, 370
- Villaret and Klippel. The Asthenias and Myopathic Atrophies, 295
- Villiger Emil. Gehirn und Rückenmark (Review), 239
- Voisin, Jules, and Roger. Potassium Bromide in Epilepsy, 352
- Voisin, Roger, Jules Voisin, and A. Rendu. Idiocy and Cerebellar Lesion, 523
- Voisin, Jules, Roger Voisin, and A. Rendu. Idiocy and Cerebellar Lesion, 523
- Vorkastner. Jacksonian and Pseudo-Jacksonian Epilepsy, 768
- Voss, G. v. Hysterical Fever, 443
- Vulpus, Oscar. Failures in Transplantation of Tendons, 862
- WALKER. Dementia Præcox, 519
- Walton and Paul. Cerebral Element in Reflexes, its Relation to Spinal Element, 843
- Warrington, W. B., and John Owen. Pathology of Myelitis Acutissima Hemorrhagica Disseminata, 401; Hemorrhage into Brain and Cord from Obliterative Arterial Disease, 407
- Waterman and Putnam. Cerebellar Tumours, 510
- Wehrli. Cortical Blindness, etc., 372
- Wehrung, Gaston. Unilateral Loss and Subsequent Return of a Knee-Jerk, 515
- Weisenburg, T. H. Bulbar Symptoms with Carcinoma of Parts other than Nervous System from Intoxication, 300; Adult Hemiplegia, 366; Contractures in Organic Nervous Diseases, 375
- Weisenburg and Mills. Word Blindness, Treatment of Visual Aphasia, 152; Localisation of Higher Psychic Functions, 420; Cortical Representation of Cutaneous and Muscular Sensibility, 819
- Weisenburg and Thorington. Syringomyelia with Double Optic Neuritis, 138
- Westphal. Traumatic Hysteria resembling Ophthalmoplegia Externa 144; Malformation of Spinal Cord, 497
- Weygandt. Idiocy, 225

- |  |   |
|--|---|
| <p>White, W. A. Types in Mental Disease, 455</p> <p>Williams, Tom A. Report of Congress of French Alienists and Neurologists, 718, 797, 863</p> <p>Wilson, John Gordon. Taste-buds of Larynx, 48</p> <p>Winfield. Somatic Evidences of Syphilis in Paretics, 512</p> | <p>Workman, Charles, and Walter K. Hunter. Cord and Medulla in Acute Ascending Paralysis, 106</p> <p>Wyler, Marcus. Outlines of Comparative Lunacy Law (Review), 869</p> <p>ZBINDEN. Psychological Conception of Nervousness, 370</p> |
|--|---|











ONE DAY

1 DAY USE  
RETURN TO DESK FROM WHICH BORROWED  
Biology Library

This publication is due on the LAST DATE  
stamped below.

ILL

Med Center

SF

3 days only

Borrowed from RETURNED - Berkeley

~~OCT 16 1958~~

OCT 16 1958

OCT 28 1958

ONE DAY MAY 20 1965

MAY 20 1965

LD 23-10m-8,'57  
(C8364s10)4186

General Library  
University of California  
Berkeley

ONE DAY  
This book is issued for one-day use and is due before closing time on the following day.

If not returned on the date due, it will incur a fine of 25 cents per day until returned. If not returned by the fourth day overdue, the book will be replaced at the borrower's expense.

BIOLOGY  
LIBRARY

380727

RC321

Review

R 4  
v. 4

Permanent  
ONE DAY

UNIVERSITY OF CALIFORNIA LIBRARY

